MONTGOMERY WATSON

May 10, 2001

Ms. Joni Rhiner
US Army Corps of Engineers-Sacramento
1325 J Street 12th Floor
Sacramento, California 95814-2922

Subject: Final Remedial Investigation for Sites FTIR-32A and FTIR-39
Fort Irwin National Training Center

Dear Ms. Rhiner:

Enclosed are three copies of the Final Remedial Investigation (RI) for Sites FTIR-32A and FTIR-39 at the Fort Irwin National Training Center. The report replaces the Draft RI for Sites FTIR-32A and FTIR-39 submitted on June 5, 2000. All revisions were made in accordance with responses to comments in Appendix F.

The appropriate number of copies of this report have been forwarded to Fort Irwin, the Regional Water Quality Control Board, and the Department of Toxic Substances Control. If you should have any questions regarding the report, please do not hesitate to call me at 916-921-3555.

Sincerely,

Jacklyn Bowen

Jacklyn Bowen, P.E.
Project Manager

cc: Justine Dishart (Fort Irwin – DPW)
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PROJECT STAFF

This final report entitled "Remedial Investigation for Sites FTIR-32A and FTIR-39", located at the Fort Irwin National Training Center, California, was prepared under the direction of the U.S. Army Corps of Engineers, Sacramento District (USACE). The following key project staff with the USACE and Montgomery Watson provided technical leadership and review.

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Jacklyn Bowen, P.E.

FINAL REMEDIAL INVESTIGATION FOR
SITES FTIR-32A (LOWER GOAT MOUNTAIN LANDFILL) AND FTIR-39

FORT IRWIN NATIONAL TRAINING CENTER

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ABBREVIATIONS AND ACRONYMS

ATSDR	Agency for Toxic Substance and Disease
BRAs	baseline risk assessments
BRAC	base realignment and closure
BTEX	benzene, toluene, ethylbenzene, xylenes
BUTL	background upper tolerance limit
Cal-EPA	California Environmental Protection Agency
CDFG	California Department of Fish and Game
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
cm^2	square centimeters
COPCs	Chemicals of Potential Concern
CSF	cancer slope factor
CSC	California Species of Special Concern
CSM	conceptual site model
DTSC	California Department of Toxic Substance Controls
ECAO	Environmental Criteria Assessment Office
ERA	ecological risk assessment
ESMP	Endangered Species Management Plan
FORSCOM	U. S. Army Forces Command
FS	Feasibility study
HEAST	Health Effects Assessment Summary Tables
HHERA	Human Health and Ecological Risk Assessment
HHRA	Human Health Risk Assessment
HI	hazard indices
HQs	hazard quotients
hr	hour
ILCR	incremental lifetime cancer risk
IRIS	Integrated Risk Information System
IRP	Installation Restoration Program
kg	kilogram
m	meters
m^2	square meter
m^3	cubic meter
MAAR	Mojave Anti-Aircraft Range
µg	micrograms
mg	milligrams
mg/day	milligrams per day
msl	mean sea level
NASA	National Aeronautics and Space Administration
NOAEL	no-observable-adverse-effect level
NRC	National Research Council
NTC	National Training Center
PAHs	polynuclear aromatic hydrocarbons
Parsons ES	Parsons Engineering Science

ABBREVIATIONS AND ACRONYMS
(Continued)

PCDD	polychlorinated dibenzo-p-diotins
PCDF	polychlorinated dibenzofurans
PEA	Preliminary Endangerment Assessment
PRG	preliminary remediation goals
RAGS	Risk Assessment Guidance for Superfund
RDA	recommended daily allowance
RfD	reference dose value
RI	Remedial Investigation
SARA	Superfund Amendments and Reauthorization Act
SI	Site Inspection
SVOC	semi-volatile organic compound
TCDD	tetrachlorodibenzo-p-dioxin
TEF	toxicity equivalency factor
TEQs	toxicity equivalents
TopC	Technical Operations Center
TPH	total petroleum hydrocarbons
TRPH	total recoverable petroleum hydrocarbons
UCL	upper confidence limit
USACE	United States Army Corps of Engineers
USDA	United States Department of Agriculture
USEPA	United States Environmental Protection Agency
USFWS	United States Fish and Wildlife Service
VOCs	volatile organic compounds

EXECUTIVE SUMMARY

The United States Army Corps of Engineers – Sacramento District (USACE) contracted Montgomery Watson under Contract Number DACW05-95-D-0023 Delivery Order 0037 to conduct a remedial investigation and baseline risk assessment at Sites FTIR-32A and FTIR-39

Fort Irwin encompasses an area of approximately 1,000 square miles in the Mojave Desert in San Bernardino County, California. The community of Barstow is located approximately 35 miles southwest of the installation. Site FTIR-32A, Lower Goat Mountain Landfill, is located in one of the canyons south of the Technical Operations Center in the northeastern portion of the installation. The site may have been used as an open pit dump from the 1940s through the mid-1980s. Site FTIR-39, Goldstone Lake Rocket Testing Range, is located at the southern end of Goldstone Dry Lake Playa in the western portion of Fort Irwin. The site consists of a flat area on the lake bed that is occupied by three rocket engine testing areas, associated observation facilities, and rocket storage locations.

The objectives of this remedial investigation (RI) are to:

- Characterize the nature and extent of soil contamination at Sites FTIR-32A and FTIR-39.
- Evaluate baseline human health risks from contaminants identified in soils using the 95 percent upper confidence level (UCL) on the mean of existing soil data.
- Evaluate the potential for adverse impacts of soil contaminants on ecological receptors and habitats, if present

Soil-gas samples, exploratory test pit excavations, and soil samples were collected by Montgomery Watson during the site investigation in June 1997. Based on the results of the screening level human and ecological risk evaluations in the Site Investigation Report (Montgomery Watson, 1998) further evaluation of the human health risks at Site FTIR-32A and the ecological risks at Site FTIR-39 were necessary.

1.0 INTRODUCTION

Montgomery Watson was contracted by the U.S. Army Corps of Engineers, Sacramento District (USACE) under Contract No. DACW05-95-D-0023, Delivery Order 37 to perform a remedial investigation (RI) for Sites FTIR-32A (Lower Goat Mountain Landfill), FTIR-38, FTIR-39, and FTIR-40 at the National Training Center (NTC), Fort Irwin and at the Goldstone Deep Space Communications Complex (Goldstone). Site investigations and screening level risk assessments were previously conducted at Sites FTIR-32A (Upper Goat Mountain Diesel Spill), FTIR-32A (Lower Goat Mountain Landfill), FTIR-38, FTIR-39, FTIR-40, FTIR-25E, and FTIR-32B as part of Delivery Order 25 (Montgomery Watson, 1997 and 1998). Additional investigation of Sites FTIR-38 and FTIR-40, including Phase II ecological validation studies, were recommended by the California Department of Toxic Substances Control (DTSC) and agreed to by the Army. The additional investigation methods were described in the Draft Workplan for Sites FTIR-32A, FTIR-38, FTIR-39, and FTIR-40 (Montgomery Watson, 1999). The results of these additional investigations for Sites FTIR-38 and FTIR-40 are presented in the Draft Remedial Investigation/Feasibility Study (FS) for Sites FTIR-38 and FTIR-40 (Montgomery Watson, 2001).

No additional site investigation activities were recommended for Sites FTIR-32A and FTIR-39 in the Final Site Investigation (SI) report (Montgomery Watson, 1998), or during subsequent discussions between the Army and DTSC. Therefore, this RI report describes the nature and extent of contamination for Sites FTIR-32A (Lower Goat Mountain Landfill) and FTIR-39 (Goldstone Lake Rocket Testing Range) based on the results of previous field investigations. Additionally, the results of baseline human health and ecological risk assessments (HHERAs) conducted for Sites FTIR-32A and FTIR-39 are also described in this RI report.

1.1 PURPOSE AND OBJECTIVES

The purpose of this RI report is to evaluate the potential impacts of chemicals identified in soils at Sites FTIR-32A and FTIR-39 on human health and the environment The specific objectives of this RI are to:

- Characterize the nature and extent of soil contamination at Site FTIR-32A and FTIR-39.
- Evaluate baseline human health risks from contaminants identified in soils using the 95 percent upper confidence limits (UCLs) on the mean of existing soil data.
- Evaluate the potential for adverse impacts of soil contaminants on ecological receptors and habitats, if present.

1.2 SITE LOCATION

Fort Irwin encompasses an area of approximately 1,000 square miles in the Mojave Desert in San Bernardino County, California (Figure 1-1). The community of Barstow is located approximately 35 miles southwest of the installation. Fort Irwin was originally established in 1940 as the Mojave Anti-Aircraft Range (Camp MAAR) to provide a facility where training in the use of anti-aircraft weapons could be conducted without interruption. The California Institute of Technology also used the area around Goldstone Dry Lake for rocket testing. In 1972, the California National Guard assumed operation of the facility. The U.S. Army was reissued command of the facility in 1981, and Fort Irwin became the National Training Center for the Army The installation is currently under the command of the U.S. Army Forces Command (FORSCOM).

1.2.1 Site FTIR-32A – Lower Goat Mountain Landfill

The Lower Goat Mountain Landfill is located in one of the narrow canyons south of the Technical Operations Center (TOpC) (Figure 1-2). The site may have been used as an open pit dump from the 1940s through the mid-1980s. A record search indicated that the site was used as

an unauthorized open pit dump in 1986 and 55-gallon drums, 5-gallon oil and anti-freeze cans, targets, ammunition boxes, and ration containers were removed from the site.

1.2.2 Site FTIR-39 – Goldstone Lake Rocket Testing Range

The Goldstone Lake Rocket Testing Range is located at the southern end of Goldstone Dry Lake Playa (Figure 1-2). The site consists of a flat area on the lake bed that is occupied by three rocket engine testing areas, associated observation facilities, and rocket storage locations The site was utilized by Cal Tech to develop and test rockets during the period from 1941 to the end of World War II. A variety of rockets were tested at Goldstone, including: anti-submarine rockets, barrage rockets, high-velocity aircraft rockets, spin-stabilized rockets, and British rockets During the period from 1943 until the end of World War II, rocket testing activities shifted away from Goldstone to another military facility.

1.3 REPORT ORGANIZATION

The organization of this report is as follows:

Section 1.0 Introduction

Section 2.0 Environmental Setting

Section 3.0 Previous Investigations

Section 4.0 Contaminant Fate and Transport

Section 5.0 Baseline Human Health Risk Assessment

Section 6.0 Ecological Risk Evaluation

Section 7.0 Conclusions and Recommendations

The following information is included as appendices to this report:

Appendix A	Photographs of Sites FTIR-32A and FTIR-39
Appendix B	Technical Memoranda
Appendix C	Toxicology Profiles
Appendix D	Human Health Risk Assessment Calculations
Appendix E	Baseline Risk Assessment for Hypothetical Future Residents
Appendix F	Responses to Comments

2.0 ENVIRONMENTAL SETTING

The physical and biological resources associated with NTC Fort Irwin and Goldstone in general, and Sites FTIR-32A and FTIR-39 specifically, are presented in this section.

2.1 FACILITY DESCRIPTION

Site FTIR-32A (Lower Goat Mountain Landfill) is located within the boundaries of NTC Fort Irwin, and is used as a range for firing and training maneuvers. Site FTIR-39 (Goldstone Lake Rocket Testing Range) is located within Goldstone at the southern end of the Goldstone Dry Lake Playa. The site was used to test rockets during the period from 1941 to the end of World War II. Rockets are no longer tested at Site FTIR-39, and the site is now part of the National Aeronautics and Space Administration (NASA) Deep Space Satellite Tracking Station

2.2 GEOLOGY, HYDROGEOLOGY, AND SURFACE WATER HYDROLOGY

Site FTIR-32A (Lower Goat Mountain Landfill) is characterized by a thin veneer of soil (5 to 15 feet thick) overlying bedrock. No aquifers are expected to immediately underlie this area. Geotechnical soil samples collected from Site FTIR-32A indicated that soils at this site consist of clayey sand with gravel. The site is underlain by granite. Jointed granitic bedrock may have limited open fractures available for flow, but overall permeabilities are expected to be extremely low and decrease to zero with increasing depth Therefore, site bedrock is expected to act as an impermeable barrier, and any flow that does occur is likely to be transient. Local surface runoff is towards the south and west. The site lies at the eastern boundary of the watershed that drains into Drinkwater Dry Lake, three miles to the west The groundwater basin that is associated with this watershed is referred to as the Avawatz Groundwater Basin; however, there are no known wells in the vicinity of the site or in the Avawatz Valley Groundwater Basin.

Site FTIR-39 is located in and on the southern edge of the Goldstone Dry Lake Playa. The site is underlain by fine-grained, lacustrine sediments. Geotechnical testing indicated that surface soils

at Site FTIR-39 consist of silty sand. The thickness of alluvial and lacustrine sediments underlying this site is unknown, but is expected to exceed several hundred feet as indicated by the logs of well borings drilled in the vicinity [Parsons Engineering Science (Parsons ES), 1995]

2.3 BIOLOGICAL RESOURCES

An overview of the biological resources at NTC Fort Irwin was provided by Mickey Quillman, Natural and Cultural Resources Manager for NTC Fort Irwin, and is the basis for the descriptions provided in the following subsections.

2.3.1 Vegetation

A detailed list of plants occurring at NTC Fort Irwin is presented in Table 2-1. The most common vegetation associations in the vicinity of NTC Fort Irwin are creosote bush scrub and saltbush scrub. Creosote bush scrub is the most widespread community on the NTC, occurring throughout the range below 3,600 feet (1,100 meters [m]) on alluvial slopes, valley floors, and mountain slopes (Gibson et al., 1994). A sub-association of this vegetation type is described as the creosote-burrobush association based on the widespread dominance of the creosote bush (*Larrea tridentata)* and burrobush (*Ambrosia dumosa)*. Burrobush is a much smaller shrub that may often be more abundant than creosote bush, but the projected foliar cover and volume is generally less than that provided by the creosote bush. Many subdominant shrubs occur in creosote bush scrub. These include range ratany (*Krameria erecta)*, silver cholla (*Opuntia echinocarpa)*, Anderson's boxthorn (*Lycium andersonii),* desert straw (*Stephanomeria pauciflora*), wishbone bush (*Mirabilis bigelovii*), and cheesebush (*Hymenoclea salsola*). At higher elevations, subdominant species include California buckwheat (*Erigonum faciculatum*), hopsage (*Grayia spinosa*), winter fat (*Krasheninnikovia lanata*), and bladdersage (*Salazaria mexicana*).

Saltbush scrub is characterized by the dominance of one or more species of saltbush. Saltbush scrub is associated with moderately alkaline soils toxic enough to inhibit most desert shrubs that occur in the creosote bush scrub. It commonly occurs on lower bajada slopes and plains, and

around playas throughout most of the deserts (Quillman, 1996). Good examples of saltbush scrub can be found on the playas along the margins of dry lakes on the NTC. Common shrubs are shadscale (*Atriplex confertifolia*), Mojave saltbush (*A. spinifera*), four-winged saltbush (*A. canescens*), and allscale (*A. polycarpa*). Other shrubs found in association with saltbush scrub include budsage (*Artemisia spinecens*), winterfat, hopsage, and Anderson's boxthorn. Typically, one strongly dominant species of saltbush is found in association with a smaller number of saltbush species in a particular area. Russian thistle (*Salsola tragus*), commonly known as tumbleweed, can often be found in saltbush scrub, especially in sandy areas. A large, dense stand of this species occurs in the southwest portion of Langford Dry Lake.

Other Mojave Desert plant associations occur at NTC Fort Irwin, but are less common at the subject sites. These associations are:

- Blackbush Scrub
- Mojave Mixed Woody Scrub
- Mojave Desert Wash Scrub
- Alkalai Sink Scrub
- Seeps and Springs vegetation
- Joshua Tree Woodland
- Juniper Woodland

2.3.2 Wildlife

The wildlife species found at NTC Fort Irwin are presented in Tables 2-2 through 2-4. Although Fort Irwin and the surrounding desert appear uniform, the desert supports a variety of wildlife habitats. Wildlife habitats are described by the vegetation types that occur in a particular area. Fort Irwin consists primarily of creosote bush scrub habitat. While each vegetation type identified previously (Section 2.3.1) contains similar faunal components, there are species that occur more abundantly or solely in certain habitat types. For example, the zebra-tailed lizard (*Callisaurus draconoides*) occurs in nearly all vegetation communities on the NTC, but is more

common in desert washes; the common night lizard (*Xantusia vigilis*) occurs almost exclusively in Joshua tree woodland.

Most wildlife species that occur on the NTC are adapted to desert scrub habitats that provide little cover and xeric (i.e., dry) conditions. However, seeps and springs provide perennial sources of water and a high concentration of vegetation and cover that increase wildlife diversity in these areas. Large mammals such as bighorn sheep, coyote, and desert kit fox know the locations of these water sources and return to them regularly; bats typically forage over these areas because of the increased abundance of invertebrate prey. The continued use of springs by wild burros has resulted in highly disturbed areas that now require management. Bird species that migrate in the spring and fall (and are not usually associated with desert environment) may stop over in these areas to forage and rest during their migration.

Areas of rocky terrain, such as the Avawatz, Granite, and Tiefort mountains, as well as other mountainous and hilly ranges onsite, provide suitable habitat for many reptiles, rodents, and bird species. Along with the different vegetation communities that normally occur as one ascends these ranges, differences in slope, aspect, and elevation result in microhabitats that support different wildlife species. Notable species that occur in these areas include bats, which rely on rocky outcrops for roosting sites, and raptors, which use cliff faces and rocky ledges in mountain habitat as sites to roost or nest.

Several dry lakes occur on the NTC. These areas provide little wildlife habitat because they are basically devoid of vegetation. However, they do contain algae that support brine shrimp and form the base of a food chain. Migratory waterfowl and large mammals may visit these areas after periods of heavy rainfall.

As is typical of most desert systems, larger animal species are uncommon, widely dispersed, and often nocturnal. Smaller mammals and reptiles, highly adapted to desert conditions, are much more common, but are often either secretive, nocturnal, or active for only short periods of the year. Birds are among the most conspicuous species, usually occurring in greatest concentrations in the vicinity of washes and springs where more structured and complex

vegetative assemblages occur. With some exceptions, wildlife (such as birds and larger mammals) is generally more mobile and not limited to a single habitat type. Therefore, it should be noted that the entire NTC is likely used in the course of an organism's daily or seasonal activity patterns, particularly for larger and/or more mobile species. It should also be noted that some species on site (e.g., fish, amphibians, and some reptiles and mammals) are highly adapted for one habitat type and restricted to these areas. Lack of these habitats might have contributed to the absence of native amphibians and fish populations on the installation.

Although wildlife surveys typically do not focus on invertebrate species, invertebrates are an essential component of desert ecosystems, providing food for numerous vertebrate species and acting as pollinators for a large number of plant systems. The seasonal reproductive cycle of some insect species results in an "explosion" of the population in a relatively short period of time. This "swarming" of individuals provides an important prey base for insectivores, such as smaller birds, reptiles, amphibians, and bats.

Work begun on the NTC in 1995 by Pratt suggests that high levels of invertebrate diversity can be found in isolated areas. Because the diversity of insects is often correlated with the diversity of plants in an area, the springs on the NTC are particularly important to the invertebrate populations. The Avawatz mountains above 4,000 feet mean sea level (msl) exhibit high levels of endemism for a number of insect species (Pratt, 1996). The only known population of one subspecies of the square-spotted blue butterfly (*Euphilotes battoides ellisi*) occurs there.

The following section summarizes, by taxon, the biological diversity of Fort Irwin. Each vertebrate taxonomic group is addressed.

2.3.2.1 Fishes. Although numerous active perennial springs are located in the study areas, no documentation exists of native fish species occurring in any of these springs. Non-native species, such as the mosquitofish (*Gambusia affinis*), occur in some ponds to control mosquitoes. No other native or non-native fish species are known to occur in any spring on the NTC.

2.3.2.2 Amphibians. No amphibians have been observed on Fort Irwin; however, any of the active springs could potentially support amphibian species, even springs that are active only part of the year. One amphibian species, the red-spotted toad (*Bufo punctatus*), is likely to occur on the NTC. The red-spotted toad is a widespread species that occurs in a variety of habitats, including desert oases and springs. However, it has not been observed on the base to date.

2.3.2.3 Reptiles. The rich, diverse reptilian populations known to occur on the NTC are characteristic of those found in creosote scrub habitat (Table 2-2). Some diurnal (i.e., active during the day) lizards are widespread, while others are habitat specialists. Widespread and abundant species include zebra-tailed lizards (*Callisaurus draconoides*), side-blotched lizards (*Uta stansburiana*), and western whiptails (*Cnemidophorus tigris*). Other lizard species that are widespread but less abundant include the desert horned lizard (*Phrynosoma platyrhinos*), long-nosed leopard lizard (*Gambelia wislezenii*), and the desert iguana (Dipsosaurus dorsalis). Habitat specialists include collard lizard (*Crotaphytus insularis*), chuckwalla (*Sauromalus obesus*), sagebrush lizard (*Urosaurus graciosus*), and common night lizard (Morofka, 1993). There are two populations of Mojave fringe-toed lizard (*Uma scoparia*) on the NTC. The main population is found in the dunes just north of Bitter Spring. The other population is found in the dunes just east of Red Pass Dry Lake.

Some of the more common snake species occurring at NTC Fort Irwin include the coachwhip (*Masticophis flagellum*), gopher snake (*Pituaphis melanoleucus*), western patch-nosed snake (Salvadora hexalepis), western shovel-nose snake (*Chionactis occipitalis*), and the side-winder (*Crotalus cerastes*) (Quillman, 1996; Chambers Group, 1992b). Less common species include the blind snake (*Leptotyphlops humulis*) and ground snake (*Sonora semiannulata*). Unlike the lizards, most of which are primarily diurnal, most of the snake species that occur on the NTC are nocturnal.

The desert tortoise (*Gopherus agassizii*) is known to occur throughout the NTC in low to moderate numbers, with the highest concentration along the southern boundary (Chambers Group, 1996a). Numerous surveys have been conducted over the past years to document the distribution and estimated size of tortoise populations throughout the NTC (Chambers Group,

1994; Krzysik and Woodman 1991; Woodman, and Goodlet, 1990). The U.S. Fish and Wildlife Service (USFWS) determined that the desert tortoise warranted listing in response to documented population declines over large portions of its range. The decline is thought to be due to a number of reasons, including upper respiratory tract disease exacerbated by the stress of several drought seasons, loss of habitat, predation by ravens, livestock grazing, and direct disturbance by humans. The USFWS emergency-listed the desert tortoise on 4 August 1989, and officially listed the Mojave population as federally threatened in April 1990 (Quillman, 1996).

The NTC Fort Irwin has adopted a series of programs intended to benefit the desert tortoise. Each program undertaken on behalf of the desert tortoise at Fort Irwin contributes to a better understanding of the species and the conservation and preservation of the species as a whole. These programs include education programs for military and civilian personnel, juvenile tortoise research, reconnaissance-level surveys for the tortoise as well as other general sensitive plant and wildlife species, and long-term studies that include desert tortoise monitoring plots, tortoise relocation, upper respiratory tract disease, neonatal information, and desert tortoise predation (see Chambers Group, 1996a for a full description of desert tortoise programs).

2.3.2.4 Birds. Most of the bird species that occur on the NTC are representative of creosote bush scrub habitat (Table 2-3). Some of the more common bird species include black-throated sparrow (*Amphispiza bilineata*), rock wren (*Salpinctes obsoletus*), horned lark (*Eremophila alpestris*), common raven (*Corvus corax*), and greater roadrunner (*Geococcyx californiaus*). The verdin (*Auriparus flaviceps*) and black-tailed gnatcatcher (*Polioptila melanura*) are more common in desert wash systems.

The greatest bird activity, as is true in most habitats, is concentrated in the immediate vicinity of water. On the NTC, springs are a valuable resource to most resident and migratory bird species. Not only is there increased structural diversity of the vegetation and habitat, invertebrate species that become abundant in the vicinity of the springs during the spring and summer provide an important food source to resident species. Representative birds include house finch (*Carpodacus mexicanus*), the flycatcher phainopepla (*Phainopepla nitens*), Northern mockingbird (*Mimus polyglottos*), and song sparrow (*Melospiza melodia*). Numerous birds occur as winter or summer

residents, or migrants that occur only during brief periods in the spring and fall. Some of the more common representative species include yellow-rumped warbler (*Dendroica coronata*), Hutton's vireo (*Vireo huttoni*), cliff swallow (*Hirundo pyrrhonata*), ruby-crowned kinglet (*regulus calendula*), and white-crowned sparrow (*Zonotrichia leucophrys*).

Red-tailed hawks (*Buteo jamaicensis*), northern harriers (*Circus cyaneus*), golden eagles (*Aguila chrysaetos*), and prairie falcons (*Falco mexicanus*) are some of the raptors that occur on the NTC. Many raptor species use the cliff faces and rocky ledges of mountain ranges as sites to roost or nest. Owl species include the burrowing owl (*Speotyto cunicularia*), barn owl (*Tyto alba*), and short-eared owl (*Asio flammeus*)

2.3.2.5 Mammals. Several mammalian species are known to occur on the NTC (Table 2-4). The majority of desert mammals are nocturnal, but a few may be seen by day. Small mammals most frequently observed throughout the NTC include blacktail jackrabbit (*Lepus californicus*) and white-tail antelope squirrel (*Ammospermophilus leucurus*). Common rodent species include the desert kangaroo rat (*Dipolomys deserti*), Merriam's kangaroo rat (*D. merriami*), long-tailed pocket mouse (*Perognathus formosus*), little pocket mouse (*P. longimembris*), and desert woodrat (*Neotoma lepida*) (Quillman, 1996). The Mojave ground squirrel (*Spermophilus mojavensis*) also occurs within the NTC.

Larger mammal species occurring at the NTC include the badger (*Taxidea taxus*), kit fox (*Vulpes macrotis*), gray fox (*Urocyon cinereoargenteus*), coyote (*Canis latrans*), bobcat (*Lynx rufus*), and mountain lion (*Felis Concolor*). The kit fox and coyote are expected to occur throughout the NTC, whereas the others are localized and fairly rare. Another large mammal is Nelson's bighorn sheep (*Ovis canadensis nelsoni*), a fully protected sensitive species

Mines and natural caves located throughout the NTC provide potential roosting habitat for bats. Bats also use the many cliff faces and rocky ledges of the mountain ranges in the study area as sites for roosting. Seven bat species were detected on the NTC during recent surveys by Brown (1994). The western pipistrelle (*Pipistelle hesperus*) and California myotis (*Myotis californicus*) were the two most commonly observed species.

2.3.3 Sensitive Species

The NTC Fort Irwin provides habitat for certain sensitive species Sensitive plant species include all listed federal or state threatened, endangered, or otherwise sensitive species, and species considered to be rare or declining by the California Native Plant Society. Sensitive wildlife species include all listed federal and state threatened and endangered species, species that are candidates for such listing, and California Species of Special Concern (CSC).

An Endangered Species Management Plan (ESMP) is currently being prepared for all listed endangered, threatened, or otherwise sensitive wildlife and plant species on the NTC (Chambers Group, in preparation). In addition, a Programmatic Management Plan (PMP, Chambers Group, 1996a) for the desert tortoise was prepared to guide the management of the desert tortoise at the NTC. These documents are to be used as a guide for the continued preservation and management of the desert tortoise and other sensitive species and their habitats within the NTC.

2.3.4 Site FTIR-32A - Lower Goat Mountain Landfill

The area surrounding Site FTIR-32A is located in creosote scrub habitat, with the dominant perennial plants being creosote bush, burro bush, desert trumpet and cheese bush. Typical wildlife in the general vicinity of Site FTIR-32A include the more common birds, lizards, and ground squirrels. However, the habitat at Site FTIR-32A has been altered substantially (Mickey Quillman, Natural and Cultural Resources Manager for NTC Fort Irwin). A biological field reconnaissance conducted by representatives of DTSC and the California Department of Fish and Game (CDFG) on March 23, 1999 biologists to conclude that the majority of the site is devoid of vegetation and provides inadequate habitat for the Mojave ground squirrel, a primary indicator species evaluated in the quantitative screening ecological assessment in the SI report (Montgomery Watson, 1998). Based on the low quality of habitat at Site FTIR-32A, the Army with DTSC and Department of Fish and Game concurrence decided not to investigate this site further in regard to ecological concerns. The implications of this decision are described further in Sections 6.0 and 7.0.

2.3.5 Site FTIR-39 - Goldstone Lake Rocket Testing Range

Vegetation in the vicinity of Goldstone is dominated by the saltbush species, *Atriplex confertifolia*, and *A. polycarpa*, with burro bush, golden head and a few creosote bushes. However, Site FTIR-39 is located at the southern end of the Goldstone Dry Lake Playa, a large, flat salt-pan that is almost entirely devoid of vegetation. The site has been identified by Mickey Quillman (Natural and Cultural Resources Manager for NTC Fort Irwin) as providing inadequate habitat for the Mojave ground squirrel (personal communication between Mickey Quillman and Ms. Tiffany Gates-Tull, U.S. Army, March 11, 1999). In addition, during a visit to Site FTIR-39 DTSC and the Department of Fish & Game agreed that there is insufficient habitat at the site for the Mojave ground squirrel. Additionally, Site FTIR-39 contains no sensitive plant species and the desert tortoise is not present at the site.

3.0 PREVIOUS INVESTIGATIONS

Descriptions of Sites FTIR-32A and FTIR-39, and the previous investigations conducted for these sites, are provided in the following subsections. Brief summaries of the site history for each site were generally excerpted from the Parsons ES Workplan (Parsons ES, 1995). Additional information from Montgomery Watson's site reconnaissance, performed in 1997, is included where appropriate. Photographs of Sites FTIR-32A and FTIR-39 are included in Appendix A

3.1 SITE FTIR-32A – LOWER GOAT MOUNTAIN LANDFILL

The historical uses of Site FTIR-32A – Lower Goat Mountain Landfill are described in Section 3.1.1. The previous investigations that have been conducted at the site are presented in Section 3.1.2.

3.1.1 Site Description

Site FTIR-32A, Lower Goat Mountain Landfill, is located in one of the narrow canyons south of the TOpC. As shown in Figure 3-1, the site covers an area of approximately 600 feet by 100 feet, and is comprised of two specific disposal areas (Parsons ES, 1995). Area 1 contained minor amounts of metallic debris and covers an area 400 feet by 100 feet within an alluvial wash. Area 2 is located a few hundred feet north of Area 1. Landfill debris noted in Area 2 during the SI in June 1997 consisted of highly rusted metal debris including concertina wire, engineer's stakes, artillery rounds, shell casings, tear gas canisters, and various types of mines (including training mines, anti-tank mines, and smoke pots). The site may have been used as an open pit dump from the 1940s through the mid-1980s. A records search indicated that the site was used as an unauthorized open pit dump in 1986. The site was found to contain 55-gallon drums, 5-gallon oil and anti-freeze cans, targets, ammunition boxes, and ration containers.

3.1.2 Previous Investigations Conducted at Site FTIR-32A

Soil-gas samples, exploratory test pit excavations, and soil samples were collected by Montgomery Watson at Site FTIR-32A during the SI conducted in June 1997 (Figure 3-1) (Montgomery Watson, 1997). A burial pit containing metal debris and some burned material was found at Site FTIR-32A. Only minor surface debris was identified in all other portions of Site FTIR-32A. Soil samples collected in the vicinity of the burial pit contained detections of volatile organic compounds (VOCs) and semi-volatile organic compounds (SVOCs) as well as elevated levels of metals. The compounds that were determined to pose a potential ecological risk in the SI are presented on Figure 3-1. The results of the soil-gas samples, exploratory test pit excavations, and soil samples are presented in the Final Data Summary Report for SI of Seven Sites: FTIR-32A (Lower Goat Mountain Landfill), FTIR-32A (Upper Goat Mountain Diesel Spill), FTIR-32B, FTIR-25E, FTIR-38, FTIR-39, and FTIR-40 (Montgomery Watson, 1997). The soils data for Sites FTIR-32A and FTIR-39 are summarized in Section 5.2 of this RI report.

A screening human health risk evaluation was conducted for Site FTIR-32A, and the results were presented in the SI Report (Montgomery Watson, 1998). Screening cancer risk and noncancer hazard estimates based on maximum concentrations of chemicals detected in surface and subsurface soils exceeded risk and hazard criteria for unrestricted (i.e., hypothetical future residential) land uses. Subsurface soils for this site also exceeded the generally accepted risk criterion for future industrial land uses. The estimated noncancer hazard index (HI) of 3.2 for a current military worker potentially exposed to surface soils slightly exceeded a hazard criterion of 1.0 (Montgomery Watson, 1998).

A screening-level quantitative ecological risk evaluation was also performed for Site FTIR-32A (Montgomery Watson, 1998). Ecological HI estimates for the Mojave ground squirrel were between 1 and 10, indicating a 'limited potential' adverse effect. Ecological HI estimates for the golden eagle were less than 1.0, indicating that site soils are not anticipated to pose a hazard to upper trophic level species (Montgomery Watson, 1998). The metals posing a potential ecological hazard are presented in Figure 3-1.

3.2 SITE FTIR-39 – GOLDSTONE LAKE ROCKET TESTING RANGE

The physical characteristics of Site FTIR-39 – Goldstone Lake Rocket Testing Range and its historical use are described in Section 3.2.1. The previous investigations that have been conducted at the site are presented in Section 3.2.2.

3.2.1 Site Description

The Goldstone Lake Rocket Testing Range is located at the southern end of Goldstone Lake Playa. The site consists of a flat area on the lakebed that is occupied by three rocket engine testing areas, associated observation facilities, and rocket storage locations (Figure 1-2). The testing areas consist of concrete pads, brackets, and/or walls that were used for securing rocket engines and for deflecting rocket exhaust. The site was utilized by Cal Tech to develop and test rockets during the period from 1941 to the end of World War II. The propellant in the rockets was ballistite, a solid powder made from approximately equal parts of nitroglycerin and nitrocellulose. No explosives were allowed on the range at Goldstone during the rocket testing years; the rocket heads were filled with plaster. Rockets were fired from the ground and from aircraft. A variety of rockets were tested at Goldstone, including anti-submarine rockets, barrage rockets, high-velocity aircraft rockets, spin-stabilized rockets, and British rockets. During the period from 1943 until the end of World War II, rocket-testing activities shifted away from Goldstone to another military facility.

3.2.2 Previous Investigations Conducted at Site FTIR-39

Site FTIR-39 was divided into three subsites, Areas 1 through 3, based on the three rocket testing facilities located in the area (Figure 3-2). Areas 1 and 3 were rocket-testing stands in which rockets were held stationary and allowed to burn in place. Area 2 was a rocket launching pad. Three surface soil samples were collected around the base of each of these rocket-testing areas in June 1997 (Figure 3-2) and analyzed for metals and nitroglycerin.

Several metals (cadmium, copper, and zinc) were detected in surface soil samples collected from Site FTIR-39 at concentrations above background levels. Explosives were not detected in any soil samples collected from the site. The results of soil sampling can be found in the Final Data Summary Report (Montgomery Watson, 1997).

A screening human health risk evaluation was conducted for Site FTIR-39, and the results were presented in the SI Report (Montgomery Watson, 1998). Screening cancer risk and noncancer hazard estimates for Site FTIR-39 were below generally accepted risk and hazard criteria for unrestricted (i.e., residential) and industrial land uses. Based on these screening risk results, no further action was recommended for Site FTIR-39, in regard to human health concerns (Montgomery Watson, 1998).

A quantitative screening ecological risk evaluation was also performed for Site FTIR-39 (Montgomery Watson, 1998). Ecological HI estimates for the Mojave ground squirrel were between 1 and 10 indicating a 'limited potential' for adverse effects. Ecological HI estimates for the golden eagle were less than 1.0, indicating that site soils are not anticipated to pose a hazard to upper trophic level species (Montgomery Watson, 1998). The metals posing a potential ecological hazard at Site FTIR-39 are presented in Figure 3-2. After completion of the Phase I ERA for Site FTIR-39, NTC Fort Irwin Biologist Mickey Quillman prepared a memorandum stating that this site contains no suitable habitat for the Mojave ground squirrel. This memorandum is included in Appendix B. Observations on the low quality habitat and the resulting implications for potential ecological concerns are described further in Sections 6.0 and 7.0 of this RI report.

4.0 CONTAMINANT FATE AND TRANSPORT

This section evaluates the effects of site-specific conditions on the probable environmental fate of each of the constituents detected in the soils at Sites FTIR-32A and FTIR-39.

4.1 METALS

Metals were detected in soil samples at concentrations above background levels (refer to Section 5.2). While specific reactions for individual metals vary slightly, the primary process affecting the fate of all metals is sorption. Most metals are precipitated, complexed with iron oxides, and sorbed onto soils soon after introduction to the environment. Because these metals are strongly sorbed onto soils, they are not expected to travel significant vertical distances through the subsurface or into groundwater. Because there are no known degradation reactions for most metals, they are expected to persist in soil.

4.2 VOLATILE ORGANIC COMPOUNDS

Tetrachloroethylene, toluene, and trichloroethylene were detected at Site FTIR-32A Lower Goat Mountain Landfill. The primary fate of VOCs in soils is volatilization, especially in arid regions, which may explain why VOCs were not detected in other soil samples collected from this site.

4.3 SEMI-VOLATILE ORGANIC COMPOUNDS

Low levels of SVOCs, including polycyclic aromatic hydrocarbons (PAHs) and phthalates, were detected in subsurface soil samples collected from Site FTIR-32A. While phthalates are commonly found as sampling or laboratory artifacts, they may also be present in soils due to leaching/diffusion from disposed plastics. PAHs and phthalates have very low Henry's Law constant values, which limits volatilization. Thus sorption and biotransformation are the primary processes controlling these SVOCs in soil/water environments. SVOCs are also highly sorbed to soils. While degradation is expected to be slower than normal due to the limited available

moisture, this process is expected to be the primary fate of the SVOCs detected at the site. The half-lives for transformation of phthalates, for example, are expected to be on the order of years.

4.4 DIOXIN/FURANS

Various dioxin/furan congeners were detected in subsurface soil at FTIR-32A. However, these concentrations were well below those contributing to significant risk in the screening risk assessment (Montgomery Watson, 1998). Dioxins/furans are generally considered to be relatively immobile in soils.

4.5 TOTAL PETROLEUM HYDROCARBONS

Petroleum hydrocarbons were detected in soil samples collected from Site FTIR-32A Lower Goat Mountain Landfill, however, VOCs that are typically associated with petroleum hydrocarbons were not detected at these sites. The petroleum hydrocarbons detected in the soils are most likely the longer-chain, less-soluble compounds. These compounds tend to sorb to the soil with only the more soluble constituents dissolving into the soil water. Biotransformation may also be important for the surface soils contaminated with petroleum hydrocarbons.

In summary, metals, SVOCs, dioxin/furans, and total petroleum hydrocarbons (TPH) are considered to be relatively immobile in desert soils. Only VOCs which were detected in Site FTIR-32A surface and subsurface soils are considered to be significantly mobile. However, these compounds were detected in only a few samples and at low concentrations. None of the other chemicals at Sites FTIR-32A and FTIR-39 are considered to be mobile. Groundwater is either absent or found at depths greater than 500 ft in the vicinity of Sites FTIR-32A and FTIR-39. Therefore, the probability of constituents detected in site soils migrating to groundwater is unlikely. The primary mechanism of transport for compounds that are highly sorbed to soils is surface water runoff, which is limited in this extremely arid environment.

5.0 BASELINE HUMAN HEALTH RISK ASSESSMENT

The methods and results of the baseline human health risk assessment (HHRA) conducted for Sites FTIR-32A and FTIR-39 are presented in this section. These assessments were conducted in compliance with the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) Remedial Response process, as amended by the Superfund Amendment and Reauthorization Act (SARA), integrated with Installation Restoration Program (IRP) requirements. The goal of the Remedial Response Process is to coordinate and conduct remedial actions as necessary to protect human health and the environment from releases of hazardous substances. The HHRA is intended to provide an analysis of the existing and potential risks that may be posed to human health by contaminants present in site media. The results of the HHRA provide the basis for determining the levels of chemicals that can remain on site and still be protective of public health. Additionally, the results of this HHRA, in conjunction with the results of the ecological risk assessment (ERA), will support the evaluation of a no-action alternative, or further action including the potential evaluation of remedial alternatives for each identified source area.

5.1 INTRODUCTION

The baseline HHRA presented in this report was conducted according to the risk assessment methodologies described in the approved *Project Workplan for the SI and RI of 31 Sites at the NTC Fort Irwin, California* (Parsons ES, 1995), hereafter referred to as the Workplan. All validated chemical data that were obtained during previous site investigations were used for the baseline HHRA; the data validation reports for these analytical results are contained in the Final Data Summary Report (Montgomery Watson, 1997).

A screening HHRA was previously conducted for Sites FTIR-32A and FTIR-39 (Montgomery Watson, 1998). Screening cancer risk or non-cancer hazard estimates for FTIR-32A surface soils exceeded screening criteria for hypothetical future residents and military personnel. Screening risk estimates for Site FTIR-32A subsurface soils exceeded screening risk criteria for future

industrial workers. Excess screening risk estimates were primarily associated with the presence of arsenic and manganese in surface soils, and arsenic and dioxins/furans in subsurface soils. Screening cancer risk and non-cancer hazard estimates for Site FTIR-39 were below screening risk and hazard criteria. Therefore this site was proposed for no further action in regard to human health concerns (Montgomery Watson, 1998)

The screening HHRA for Site FTIR-32A was conducted based upon assumptions regarding unrestricted (i.e., residential) future land use and maximum exposure point concentrations, consistent with DTSC's *Recommended Outline for Using Environmental Protection Agency Region IX Preliminary Remediation Goals in Screening Risk Assessments at Military Facilities* (California Environmental Protection Agency [Cal-EPA], 1994b). However, the baseline HHRA presented in this RI report evaluates risks based upon more realistic assumptions relative to land use and exposures. As such, the baseline HHRA provides a more realistic evaluation of potential human health impacts. Sites for which the cumulative cancer risk is less than 1.0×10^{-6} and the cumulative noncancer HI is less than 1.0 in the baseline HHRA are generally considered for no further action in regard to human health concerns (USEPA, 1991a). Sites for which the cumulative cancer risk is between 1.0×10^{-6} and 1.0×10^{-4} may be considered for no further action, depending upon site-specific considerations including current and potential future land uses. Sites that are associated with a cumulative cancer risk or noncancer HI greater than these criteria are generally considered for further action including potential evaluation of remedial alternatives (USEPA, 1991a)

5.1.1 Purpose and Objectives

The purpose of this baseline HHRA is to provide a quantitative and qualitative evaluation of the potential human health risks associated with exposures of human receptors to chemicals identified in soils at Site FTIR-32A. Consistent with the approved Workplan (Parsons ES, 1995), baseline human health risks were evaluated for future industrial exposures. In addition, a 'current military worker' scenario was evaluated for Site FTIR-32A, because this site is within the NTC Fort Irwin range and military training exercises are routinely conducted there (refer to

Sections 5.3.2 and 5.3.3). Potential migration of soil contaminants to groundwater, and the possible human health impacts associated with groundwater contamination, were not evaluated in this baseline HHRA. As described in Sections 2.2 and 4.0, no aquifers are expected to underlie Site FTIR-32A. Therefore, groundwater pathways were not evaluated in this RI report.

The specific objectives of this baseline HHRA were to:

- Identify chemicals of potential concern (COPCs) for surface and subsurface soils
- Identify appropriate land uses and potentially exposed receptors.
- Evaluate completed exposure pathways for each receptor.
- Estimate exposure point concentrations based on the 95 percent upper confidence limit on the mean of existing soils data.
- Calculate cumulative baseline cancer risks and noncancer HIs for each receptor.

This screening HHRA was conducted in accordance with the following guidance documents and reference sources prepared by the USEPA, Cal-EPA, and the USACE:

- Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part A) (USEPA, 1989)
- Guidance for Data Useability in Risk Assessment - Interim Final (USEPA, 1990)
- Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions, OSWER Directive 9355.0-30 (USEPA, 1991a)
- Human Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors, OSWER Directive 9285.6-03, March, 1991 (USEPA, 1991b)
- Final Exposure Assessment Guidelines (USEPA, 1992)
- Preliminary Endangerment Assessment Guidance Manual (Cal-EPA, 1994a)
- Recommended Outline for Using Environmental Protection Agency Region IX Preliminary Remediation Goals in Screening Risk Assessments at Military Facilities (Cal-EPA, 1994b)
- Health Effects Assessment Summary Tables (HEAST) (USEPA, 1995)
- Risk Assessment Handbook Volume I: Human Health Evaluation (USACE, 1995a)
- Risk Assessment Handbook Volume II: Environmental Evaluation (USACE, 1995b)

- Exposure Factors Handbook (USEPA, 1997)
- Risk Assessment Guidance for Superfund (RAGS), Supplemental Guidance – Dermal Risk Assessment (USEPA, 1999a)
- USEPA Region 9 Preliminary Remediation Goals (PRGs) 1999 (USEPA, 1999b)
- Integrated Risk Information System (IRIS) database (USEPA, 2000)

5.1.2 Scope

This baseline HHRA is intended to provide a quantitative and qualitative evaluation of the potential human health risks associated with contaminants present in soils at Site FTIR-32A. Surface (0 - 1 foot bgs) and subsurface (>1 - 10 feet bgs) soil sampling data collected from Site FTIR-32A were evaluated in this baseline HHRA. As previously stated (Sections 2.2 and 4.0), aquifers are not believed to underlie Site FTIR-32A. Therefore, potential migration of contaminants to groundwater and the possible human health impacts associated with groundwater contamination were not quantitatively evaluated in this baseline HHRA (Section 5.1.1).

5.1.3 Organization of the Human Health Risk Assessment

Section 5.1 - Introduction. This section presents a brief introduction to this document and identifies the objectives and scope of the baseline HHRA.

Section 5.2 - Identification of Chemicals of Potential Concern. This section describes the methods used in the selection of COPCs for evaluation in this baseline HHRA, and summarizes the COPCs for FTIR-32A surface and subsurface soils.

Section 5.3 - Exposure Assessment. This section evaluates the current and potential future land uses for Site FTIR-32A; the current and hypothetical future human receptors potentially exposed; and the exposure pathways and assumptions used in modeling exposures for each receptor.

Section 5.4 - Toxicity Assessment. This section presents the methods used in the development of toxicity information for use in characterizing risks to each receptor.

Section 5.5 - Risk Characterization. This section presents the risk characterization methods and results of the baseline HHRA for Site FTIR-32A.

Section 5.6 - Analysis of Uncertainty. This section describes the uncertainties associated with this baseline HHRA.

5.2 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN

The chemicals identified in surface and subsurface soil samples collected from Site FTIR-32A were evaluated in a screening procedure to identify site-specific COPCs. The selection of site-specific COPCs is generally based on specific criteria, including:

- Frequency of detection
- Comparison with laboratory and field blanks
- Comparison with background concentrations
- Essential nutrient status

Each of these criteria were evaluated in the selection of surface and subsurface soil COPCs for Site FTIR-32A, as follows.

5.2.1 Frequency of Detection

As per USEPA guidance (USEPA, 1989), if data from a minimum of 20 samples of a given media are available, chemicals detected in less than 5 percent of the samples may be eliminated from consideration as COPCs in that media. If data for less than 20 samples is available, this criterion for COPC identification should not be used (USEPA, 1989). Detection frequencies for several chemicals were below 5 percent, and could have been eliminated according to the above criteria. However, it is stated in the Workplan (Parsons ES, 1995) that all chemicals detected at least once in a given medium are to be included in the quantitative risk assessment, with the exception of inorganic chemicals demonstrated to be within background levels and organic

compounds demonstrated to be laboratory contaminants Therefore, chemicals were not excluded as COPCs based on frequency of detection.

5.2.2 Comparison with Blanks

If a field sample has detectable concentrations of chemicals that are also detected in associated laboratory method blanks, trip blanks, or equipment rinsate blanks, field sample concentrations are compared to the associated blank concentrations. For chemicals commonly identified as artifacts resulting from laboratory or field procedures (e.g., methylene chloride, acetone, phthalates, etc.), the chemical detected in the field sample may not be considered to be site-related if the detected concentration is less than 10 times the blank concentration. For all other chemicals, the selection criteria used is five times the associated blank concentration (USEPA, 1990). The comparison of field sample concentrations to associated blank concentrations was performed as part of the analytical data validation task (refer to Section 2.0 of the Final SI Report). Therefore, the chemical concentrations evaluated in this risk assessment were previously evaluated by this criterion.

5.2.3 Comparison with Background Concentrations

Comparison of concentrations of chemicals detected in site media with background concentrations is appropriate for inorganic chemicals, or organic chemicals that represent 'regional' contaminants, the presence of which are not related to past site activities (USEPA, 1989). Statistical background upper tolerance limits (BUTLs) for Fort Irwin soils were previously developed by Parsons ES, as described in Section 3.0 of the Final SI Report (Montgomery Watson, 1998). The BUTLs for inorganic chemicals representative of background conditions are summarized in Table 5-1. These BUTLs were used in screening inorganic analytes as COPCs for site soils. The maximum detected concentration of each inorganic analyte was compared to its respective BUTL (Tables 5-2 and 5-3). Derivation of BUTLs was not possible for selenium and silver, due to low detection frequencies for these chemicals (refer to Section 3.2 of the Final SI Report). Therefore, concentrations of selenium and silver detected in

site soils were not screened using this criterion, and were assumed as COPCs for evaluation in the baseline HHRA.

5.2.4 Essential Nutrient Status

Calcium, iron, magnesium, potassium, and sodium are generally considered to be essential nutrients. Essential nutrients are not necessarily considered COPCs, even when media concentrations are a large fraction of what is necessary to induce a toxic response. This is because these concentrations may be beneficial, or even necessary. The following discusses nutritional requirements, typical intakes and toxic levels for these essential nutrients.

5.2.4.1 Calcium. Calcium is critical for bone formation. Other essential functions involving calcium include nerve conductions, muscle contraction, blood clotting, and membrane permeability. The recommended daily allowance (RDA) for calcium in adults is 800 milligrams per day (mg/day). For people between the ages of 11 and 24, the RDA is 1,200 mg/day; while for younger children, the RDA is between 400 and 800 mg/day depending on the age of the child. The average daily intake is 740 mg/day, and ranged from 530 mg/day in women 35 to 50 years old to 1,200 mg/day in boys 12 to 18 years old (United States Department of Agriculture [USDA], 1986; 1987). Toxic levels of calcium are not well defined. No toxic effects have been observed in many healthy adults with intakes up to 2,500 mg/day, but high intakes also induce constipation and inhibit the absorption of other essential minerals such as iron and zinc. The National Research Council (NRC) does not recommend calcium intakes much above the RDA (NRC, 1989).

5.2.4.2 Iron. Iron is a constituent of hemoglobin, myoglobin, and several enzymes. The daily iron intake in the U.S. averages 10.7 mg/day, with most iron coming from food, including vitamin-enriched foods (Murphy and Calloway, 1986). The RDA is 15 mg/day for adult women; the RDA for children, the elderly, and adult males is 10 mg/day (NRC, 1989). Adverse effects are unlikely in healthy adults with a daily intake between 25 and 75 mg. However, no data are available for the effects of doses in this range for sensitive individuals

5.2.4.3 Magnesium. Magnesium is an essential component of numerous biochemical and physiological processes. Typical magnesium intakes in the U.S. have declined from about 410 mg/day for all adults in the early 1900s (Welsh and Marston, 1982) to current levels of 330 mg/day for adult men and 210 mg/day for adult women (USDA, 1986; 1987). The RDA is 280 mg/day for adult women, 350 mg/day for adult men, and 90 mg/day for young children. Toxic levels are not well defined, but some insight can be gained from noting that antacids and laxatives such as Maalox™ and Mylanta™ generally are regarded as safe. These products each have about 200 milligrams (mg) of magnesium per teaspoon, with a normal dose of one to two teaspoons. Thus, 200 to 400 mg/day of magnesium, in addition to what is ingested in a normal diet, should be safe.

5.2.4.4 Potassium. Potassium is the principal intercellular cation in the body. Potassium also contributes to the transmission of nerve impulses, the control of skeletal muscle contractions, and the maintenance of normal blood pressure. The RDA for adults is between 1,600 and 2,400 mg/day (NRC, 1989). People who consume large amounts of fruits and vegetables have a higher potassium intake, on the order of 8,000 to 11,000 mg/day, with no apparent adverse effects (NRC, 1989).

5.2.4.5 Sodium. Sodium is the principal cation in the extracellular fluid of the body. In addition, sodium assists in regulating the membrane potential across cells. Estimates of sodium intake based on dietary surveys and analyses of urinary excretion have ranged from 1,800 to 5,000 mg/day (NRC, 1989). The range is much higher than minimum requirements. Concentrations associated with overt toxicity are not well-defined. However, chronic ingestion of high dietary sodium is associated with hypertension.

Essential nutrient status was considered in the evaluation of calcium, iron, magnesium, potassium, and sodium as COPCs for site soils (Tables 5-2 and 5-3)

5.2.5 Summary of Chemicals of Potential Concern

Chemicals detected in surface and subsurface soils collected from Site FTIR-32A were screened as COPCs for evaluation in this baseline HHRA based on the above criteria. Briefly, inorganic chemicals detected at concentrations below their respective BUTL were excluded as COPCs (Section 5.2.3). In addition, calcium, iron, manganese, potassium, and sodium were excluded as COPCs based on essential nutrient status (Section 5.2.4). Inorganic chemicals detected at concentrations greater than their respective BUTLs and all organic chemicals were included as COPCs for evaluation in the baseline HHRA, consistent with the Workplan (Parsons ES, 1995).

5.2.5.1 Site FTIR-32A. The selection of COPCs for Site FTIR-32A surface and subsurface soils is summarized in Tables 5-2 and 5-3, respectively. Chemicals selected as COPCs for surface soils (0 - 1 foot bgs) include the inorganics arsenic, barium, cadmium, copper, lead, manganese, selenium, and zinc; the VOCs tetrachloroethylene, toluene, and trichloroethylene; and total recoverable petroleum hydrocarbons (TRPH) (Table 5-2).

Chemicals selected as COPCs for subsurface soils (>1 - 10 feet bgs) include the inorganics arsenic, barium, cadmium, copper, lead, manganese, mercury, selenium, and zinc; the VOCs 1,2-dichlorobenzene, 1,3-dichlorobenzene, tetrachloroethylene, and trichloroethylene; SVOCs bis(2-ethylhexyl)phthalate, di-n-butylphthalate, hexachlorobenzene, hexachloroethane, various PAHs, and 1,2,4-trichlorobenzene; dioxins/furans; and TRPH (Table 5-3).

The COPCs selected for evaluation in the baseline HHRA for Site FTIR-32A are summarized in Table 5-4.

5.3 EXPOSURE ASSESSMENT

The results of the exposure assessment for Site FTIR-32A are presented in this section. The exposure assessment integrates information on the nature of site contaminant sources, the types of contaminants present, the receptors potentially exposed, and the potential migration and

exposure pathways available. The exposure assessment includes the development of a conceptual site model (CSM) for each source area, where appropriate. These steps are discussed below as they relate to the baseline HHRA for Site FTIR-32A.

5.3.1 Current and Future Land Uses

The NTC Fort Irwin is not scheduled for base closure, and closure is unlikely in the foreseeable future since Fort Irwin is the NTC for the U.S. Army. Site FTIR-32A exists within the boundaries of NTC Fort Irwin, and is currently part of the range area (Figure 1-2). Current and future land use designations for the range area and Site FTIR-32A under the Fort Irwin Master Plan are as a range for firing and training maneuvers. Therefore, development of residential or commercial/industrial facilities is highly unlikely in the foreseeable future. Furthermore, such facilities are most likely to occur where an infrastructure (i.e., roads, power, water supply) is already in place. Sites FTIR-32A is approximately 35 miles from the nearest city (Barstow, California). Based on the above, it is highly unlikely that this site would be developed for residential or industrial purposes under anticipated future land uses

5.3.2 Identification of Receptors

The receptors that may be potentially exposed to site contaminants were identified, based on current and potential future land uses and exposure scenarios. Under current land uses, military personnel involved in training exercises at Site FTIR-32A are the most likely receptors to be exposed to contaminants in site soils. Military personnel are also the most likely future receptor for Site FTIR-32A under anticipated future land use plans (Section 5.3.1).

For the reasons described in Section 5.3.1, it is highly unlikely that NTC Fort Irwin, or Site FTIR-32A specifically, would be converted to civilian residential or commercial/industrial land uses. It is also highly unlikely that this area of the NTC Fort Irwin would be developed for military housing. It is possible, however, that a remote testing or industrial facility could be constructed on the site in the future.

Based on the above, the potential receptors selected for quantitative evaluation in this baseline HHRA include the following:

- Current/future military personnel
- Future industrial workers

5.3.3 Evaluation of Potential Exposure Pathways

Completed exposure pathways were identified for each receptor based on anticipated land uses and site-specific conditions. Exposure of military workers to soil COPCs was assumed to occur during field exercises and maneuvers. Military personnel were assumed to be exposed to COPCs in ambient air during field training exercises. Potential exposure to soil gas infiltrating to indoor air was not assumed to be a significant pathway because indoor facilities are not currently present on Site FTIR-32A, and because climate and soil conditions preclude significant retention of VOCs in surface and near-surface soils (Parsons ES, 1995). Direct exposures of military personnel through incidental ingestion and dermal contact with soil are also anticipated to occur during field training exercises. Consistent with the Workplan (Parsons ES, 1995), military personnel were not assumed to be exposed to COPCs in subsurface soils. Based on the above, current/future military personnel may receive exposures to COPCs derived from Site FTIR-32A surface soils during field exercises and maneuvers via the following exposure pathways:

- Incidental ingestion of surface soils
- Dermal contact with surface soils
- Inhalation of volatiles and wind-borne particulates from surface soils

Hypothetical future industrial workers were also assumed to be exposed via the above pathways, but on a much more limited basis. Additionally, hypothetical future industrial workers were assumed to receive exposures to subsurface soils, because it is possible that excavation and construction activities could result in significant disturbance of subsurface soils. Potentially completed exposure pathways for the hypothetical future industrial worker include the following:

- Incidental ingestion of surface and subsurface soils
- Dermal contact with surface and subsurface soils
- Inhalation of volatiles and wind-borne particulates from surface and subsurface soils

5.3.4 Conceptual Site Model

The CSM provides a summary representation of the potentially exposed receptors and potentially complete exposure pathways. The principle components of the CSM include the following:

- Identification of the contaminant sources
- Evaluation of contaminant migration pathways
- Identification of potential receptors
- Evaluation of potential exposure pathways

A general CSM was developed for Site FTIR-32A based on the receptors selected for evaluation in Section 5.3.2, and the potentially complete exposure pathways identified in Section 5.3.3. The general CSM for Site FTIR-32A is summarized in Figure 5-1. As previously described (Section 5.3.3), completed exposure pathways were identified for surface soils, only, for current/future military personnel. Military personnel are not assumed to be exposed to subsurface soils, consistent with the exposure assumptions presented in the approved Workplan (Parsons ES, 1995).

5.3.5 Quantification of Exposures

The quantification of receptor exposures in human health risk assessment is typically based on protective assumptions relative to land use, complete exposure pathways, and calculation of exposure point concentrations. A health-protective assumption underlying all of the dose calculations is that constituent concentrations remain constant over the entire period of exposure

5.3.5.1 Exposure Point Concentration. Calculation of the exposure point concentration was based on both measured concentrations (i.e., hits) and non-detect results When a dataset contained non-detect results, one-half the sample quantitation limit was assumed for that sample. The exposure point concentrations were estimated as either the maximum or the 95 percent UCL of the arithmetic mean concentration detected in site media If the calculated 95 percent UCL of the mean concentration was greater than the maximum concentration detected, the maximum value was assumed as the exposure point concentration; otherwise the 95 percent UCL was used

The 95 percent UCL of the arithmetic mean concentration was calculated based on a lognormal distribution, according to the methods described in Gilbert (1987). Four-point Lagrangian interpolation and an H table from Gilbert (1987) were used to determine H values for use in the UCL calculation The equation for calculating the UCL of the arithmetic mean for a lognormal distribution (Gilbert, 1987) is given by:

$$UCL = e^{\bar{x}+0.5s^2+sH/\sqrt{n-1}}$$

where:

UCL = upper confidence limit
e = constant (base of the natural log, equal to 2 718)
$\bar{x}$ = mean of the transformed data
s = standard deviation of the transformed data
H = H-statistic (Gilbert, 1987)
n = number of samples

Exposure point concentrations for Site FTIR-32A surface soils are presented in Table 5-5, and those for subsurface soils are presented in Table 5-6.

5.3.5.2 Exposure Dose Calculation. The algorithms for calculating the exposure dose for each pathway are presented below In quantifying exposures associated with inhalation of volatile or particulate COPCs in air, exposure dose calculations varied between the military personnel and industrial worker scenarios Different methodologies were used for these two receptors because military personnel involved in field training and maneuvers have higher potential exposures to air contaminants than may be considered using default exposure dose calculations and assumptions (refer to Section 5.3.5.3). Inhalation exposure dose calculations for military personnel were described in the Workplan (Parsons ES, 1995), and are consistent with

methods presented in Cal-EPA (1994a). Inhalation exposure dose calculations for the industrial worker are based on standard methods described in USEPA (1989). For potential carcinogenic effects, exposure doses were averaged over a lifetime; doses for potential non-carcinogenic effects were averaged over the actual exposure period (USEPA, 1989)

For current/future military personnel and hypothetical future industrial workers, the algorithm for calculating the exposure dose due to ingestion of soil is the following:

$$Dose\ (mg/kg\text{-}day) = \frac{Cs \times IR \times EF \times ED \times UC}{BW \times AT}$$

where:

Cs = soil exposure point concentration (milligrams[mg]/kilogram[kg])
IR = ingestion rate (mg/day)
EF = exposure frequency (days/year)
ED = exposure duration (years)
UC = unit conversion (10^{-6} kg/mg)
BW = body weight (kg)
AT = averaging time (period over which exposure is averaged) (days)

For current/future military personnel and hypothetical future industrial workers, the algorithm for calculating the exposure due to dermal contact with soil is the following:

$$Dose\ (mg/kg\text{-}day) = \frac{Cs \times SA \times AF \times ABS \times EF \times ED \times UC}{BW \times AT}$$

where:

Cs = soil exposure point concentration (mg/kg)
SA = skin surface area exposed (square centimeters [cm^2]/day)

AF = soil to skin adherence factor (mg/cm^2)
ABS = absorption fraction of chemical from soil (unitless)
EF = exposure frequency (days/year)
ED = exposure duration (years)
UC = unit conversion (10^{-6} kg/mg)
BW = body weight (kg)
AT = averaging time (days)

For military personnel, only, the algorithm for calculating exposure due to inhalation of particulates from soil is the following:

$$Dose\ (mg/kg\text{-}d) = \frac{(Ca \times InhR_{normal} \times ET_{normal} \times EF \times ED) + (Ca \times InhR_{active} \times ET_{active} \times EF \times ED)}{BW \times AT}$$

where:

Ca = exposure point concentration of particulate in air (mg/cubic meters [m^3])
$InhR_{normal}$ = inhalation rate during normal activity or rest (m^3/hour [hr])
$InhR_{active}$ = inhalation rate during moderate-to-high work
ET_{normal} = exposure time engaged in normal activity or rest (hr/day)
ET_{active} = exposure time engaged in moderate-to-high work (hr/day)
EF = exposure frequency (days/year)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days)

For hypothetical future industrial workers, the algorithm for calculating exposure due to inhalation of particulates from soil is given by:

$$Dose\ (mg/kg\text{-}d) = \frac{Cs \times (1/PEF) \times InhR \times ET \times EF \times ED}{BW \times AT}$$

where:

Cs	=	exposure point concentration of particulate in soil (mg/kg)
PEF	=	particulate emission factor (m^3/kg)
InhR	=	inhalation rate (m^3/hr)
ET	=	exposure time (hr/day)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
BW	=	body weight (kg)
AT	=	averaging time (days)

The algorithm for calculating exposure due to inhalation of VOCs from soil is the following:

$$Dose\ (mg/kg\text{-}d) = \frac{(Ca \times InhR_{normal} \times ET_{normal} \times EF \times ED) + (Ca \times InhR_{active} \times ET_{active} \times EF \times ED)}{BW \times AT}$$

where:

Ca	=	exposure point concentration of VOC in air (mg/m^3)
$InhR_{normal}$	=	inhalation rate during normal activity or rest (m^3/hr)
$InhR_{active}$	=	inhalation rate during moderate-to-high work
ET_{normal}	=	exposure time engaged in normal activity or rest (hr/day)
ET_{active}	=	exposure time engaged in moderate-to-high work (hr/day)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
BW	=	body weight (kg)
AT	=	averaging time (days)

5.3.5.3 Exposure Parameters and Assumptions. The parameters and assumptions used in modeling exposure doses for the current military worker are summarized in Table 5-7. Standard, default assumptions were used in calculating daily exposure doses for the military

worker, where appropriate. However, the unique nature of this exposure scenario required the development of some exposure assumptions that deviate from default assumptions. Overall, deviation from standard, default assumptions resulted in a more health-protective exposure assessment for the current military worker than would have resulted from the use of typical exposure assumptions. The basis and rationale for the assumptions used in calculating exposures for the current military worker were presented in the Workplan (Parsons ES, 1995), and are summarized below.

Exposure point concentrations for VOCs and fugitive dust emissions in air were modeled based on maximum measured soil concentrations. In accordance with Preliminary Endangerment Assessment (PEA) guidance (Cal-EPA, 1994a), it was assumed that a chemical originating in soil is transported in air either as a volatile or as a constituent of fugitive dust, but not both Emissions of VOCs and were modeled for the current military worker using the following emission rate model described in the PEA guidance (Cal-EPA, 1994a):

$$Ca_{voc}\ (mg/m^3) = Cs_{voc}\ x\ \frac{E}{LS\ x\ V\ x\ MH}$$

where:

Ca_{voc} = VOC exposure point concentration in air (mg/m^3)
Cs_{voc} = VOC exposure point concentration in soil (mg/kg)
E = emission rate of chemical (mg/second)
LS = length dimension perpendicular to the wind (m)
V = wind speed (m/second)
MH = maximum height (m)

VOC emissions were estimated using a box model that assumed a distance of 10 meters behind a moving military tank, a MH of 6m, and a width of 3m Based on the area of the box model (10m x 3m = 30 square meters [m^2]), an estimate of 5 5m (one side of a $30m^2$ area) was assumed for

the LS. A default value for V of 2.25 m/second was assumed based on the PEA guidance (Cal-EPA, 1994a). The E was estimated using the Volatile Emission Model described in the PEA guidance (Cal-EPA, 1994a).

Fugitive dust emissions were assumed to result from wind dispersion during periods of light activity and rest, and soil disturbances from heavy vehicular traffic (e.g., military trucks or tanks) during moderate-to-high activity. A PEF of 1.6×10^7 m^3/kg for inhalation of particulates due to wind dispersion was assumed, based on a respirable particulate concentration of 61 $\mu g/m^3$ ($1/61\ \mu g/m^3 = 1.6 \times 10^7\ m^3/kg$). This respirable particulate concentration is the highest annual average value compiled over the last 4 years by the Mojave Desert Air Quality Management District in Victorville, California (Parsons ES, 1995). For estimating fugitive dust emissions associated with heavy vehicular traffic a PEF of 3.3×10^6 m^3/kg was assumed. This PEF value is equivalent to a respirable particulate concentration of 305 $\mu g/m^3$, which is approximately 5-fold higher than the highest annual average value (61 $\mu g/m^3$) compiled over the last 4 years by the Mojave Desert Air Quality Management District.

The current military worker scenario is based on the following assumptions:

- Military workers can potentially be in the field for up to 2 weeks per month for 11 months per year.
- Military workers could potentially camp in tents during each 2-week field exercise.
- Daily activities of military workers consist of 12 hrs moderate-to-heavy activity and 12 hrs light activity or rest.
- Military workers could potentially be stationed at Fort Irwin a maximum of 4 years.

Based on the above scenario, the following were assumed: an ET of 12 hr/day for light activity or rest and 12 hr/day moderate-to-high activity; an EF of 154 days per year; and an ED of 4 years. A soil IR of 75 mg/day was assumed for light activity or rest, based on a default rate of 50 mg/day (USEPA, 1997) for indoor exposures to dust (50 mg/day / 8 hrs = 6.25 mg/hr; 6.25 mg/hr x 12 hrs = 70 mg/day). A soil IR of 720 mg/day was assumed for moderate-to-high

activity, based on a default rate of 480 mg/day (USEPA, 1997) for outdoor exposures to dust (480 mg/day / 8 hrs = 60 mg/hr; 60 mg/hr x 12 hrs = 720 mg/day). An InhR of 0.83 m^3/hr was assumed for light work or rest, and an InhR of 2.5 m^3/hr for moderate-to-heavy work was assumed, consistent with USEPA default guidance (USEPA, 1997). Finally, a dermal SA of 4,300 cm^2 was assumed based on exposure of the head, arms, and hands, consistent with typical military attire and body surface area measurements (USEPA, 1999a).

The parameters and assumptions used in modeling exposure doses for hypothetical future industrial workers are summarized in Table 5-8. The majority of exposure assumptions that were used to calculate doses for hypothetical future industrial workers are based on standard default assumptions published by the USEPA (USEPA, 1989; 1997; and 1999a). The one exception is a PEF value of 1.6×10^7 m^3/kg which corresponds to the highest annual average value compiled over 4 years by the Mojave Desert Air Quality Management District in Victorville, California (Parsons ES, 1995).

5.4 TOXICITY ASSESSMENT

The toxicity assessment involves a critical review and interpretation of toxicology data from epidemiological, clinical, animal, and *in vitro* studies. The review of toxicology data ideally determines both the nature of the health effects associated with a particular chemical, and the probability that a given dose of a chemical could result in an adverse health effect. Toxicology information considered important for quantitative risk assessment includes:

- The potential for carcinogenic health effects
- The potential for chronic noncarcinogenic, adverse health effects
- The ability to cause short-term, acute effects
- The ability to affect reproduction

For carcinogens, it is assumed that no threshold dose exists, and that any dose may induce cancer. The probability of cancer development is described by the slope of the dose response

curve. The doses from various known or suspected carcinogens are assumed to be additive. For noncarcinogens, it is assumed that a dose exists below which no adverse health effects are seen (i.e., threshold dose). Compounds with short-term, acute effects are also generally considered to have a threshold dose. Compounds that affect reproduction are considered to have threshold doses unless the mechanism of action of the compound has been confirmed as one for which no threshold exists.

For purposes of conducting quantitative human health risk assessments, toxic effects of chemicals are generally categorized as carcinogenic or noncarcinogenic. The carcinogenic potential of a chemical is used in a quantitative estimate of potential cancer risk. The potential for a chemical to produce noncarcinogenic adverse health effects is used in a quantitative estimate of noncarcinogenic hazard.

5.4.1 Carcinogenic Effects of Chemicals of Potential Concern

The cancer slope factor (CSF) is the toxicity value used to quantitatively express the carcinogenic potential of cancer-causing constituents. The slope factor is expressed in units of $(mg/kg/day)^{-1}$ and represents the cancer risk per unit daily intake of carcinogenic chemical. The CSF represents the upper 95 percent confidence interval of the slope of the dose response curve. The 95 percent upper confidence interval value assures a safety factor to protect the most sensitive receptors. The product of the CSF and the exposure dose is an estimate of the risk of developing cancer from exposure to the compound of interest. Current scientific policy regards carcinogens as having additive doses and not having a threshold dose.

5.4.2 Noncarcinogenic Effects of Chemicals of Potential Concern

The reference dose value (RfD) is the toxicity value used to quantitatively express the potential for a chemical to produce noncarcinogenic effects. The RfD is expressed in units of mg/kg/day and represents a daily intake of contaminant per kilogram of body weight that is not sufficient to

cause the threshold effect of concern for the contaminant. Exposure doses that are above the RfD, or the threshold dose for noncarcinogens, could potentially cause adverse health effects.

The RfD is usually based on a no-observable-adverse-effect-level (NOAEL) derived from animal studies. An uncertainty factor is typically incorporated into the RfD, resulting in a reduction in the numerical value (i.e., resulting in a more protective toxicity value). The uncertainty factor is intended to account for uncertainties associated with (1) the extrapolation of dose-response data from animal studies to humans; (2) the existence of sensitive subpopulations within the human population; and (3) the quality of the laboratory study and database from which the dose response information is derived. Confidence in the RfD is judgmental, based on USEPA review groups and the supporting quality of the database. Chemical-specific RfDs do not account for the potential effects of chemical mixtures.

5.4.3 Pathway-Specific and Chemical-Specific Assumptions

The toxicity values used to estimate risks for current/future military personnel and hypothetical future industrial workers are presented in Table 5-9. Oral and inhalation toxicity values were generally available for most COPCs identified for Site FTIR-32A. However, the USEPA has not established toxicity values based on the dermal route of administration. For evaluating estimated exposure doses for the dermal pathway, oral toxicity values were used without modification, as specified in the approved Workplan (Parsons ES, 1995).

Following are several chemical-specific assumptions used in the toxicity assessment for this Baseline HHRA.

5.4.3.1 Cadmium. Oral or dermal CSFs are not currently available for cadmium. The available toxicology information indicates that cadmium is carcinogenic by the inhalation route of exposure. However, available data does not support a presumption that cadmium is carcinogenic by the oral or dermal routes of exposure. Therefore, the potential carcinogenic effects of cadmium were not evaluated for exposure pathways other than the inhalation route. It

should be noted that the noncarcinogenic toxicity values (i.e., RfDs) available for cadmium are based on the oral route of administration. Therefore, the potential noncarcinogenic effects attributable to exposure pathways other than inhalation (i.e., oral and dermal) were evaluated

5.4.3.2 Dioxins/Furans. The "I-TEF/89" approach was used in evaluating risks associated with dioxins and furans. The chlorine substitutions at positions 1-4 and 6-9 on either of the benzene rings of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) yield compounds commonly referred to as polychlorinated dibenzo-p-dioxins (PCDDs). Polychlorinated dibenzofurans (PCDFs) are a closely related family of compounds. There have been efforts from the scientific community to relate the toxic potency of PCDDs and PCDFs to TCDD. The most recent effort is the "I-TEF/89" method developed by USEPA in conjunction with scientists under the auspices of the North Atlantic Treaty Organization's Committee on Challenges in Modern Society

In this baseline HHRA, toxicity equivalents (TEQs) were assigned to each dioxin or furan congener based on their potencies relative to 2,3,7,8-TCDD. The TEQ was used to modify the concentration measured in soil, and the toxicity equivalent concentration for each congener was summed resulting in a total 2,3,7,8-TCDD TEQ concentration. For laboratory results reported only as total pentachloro-, hexachloro-, heptachloro-, or octachlorodibenzo-p-dioxins or furans, toxicity equivalent concentrations were not assigned. These results were not evaluated quantitatively in this screening HHRA. This is because chlorination in the 2,3,7,8-positions is required for the carcinogenic effects of PCDDs and PCDFs (USEPA, 1994a). The total 2,3,7,8-TCDD TEQ concentration was assumed as the exposure point concentration in the dose calculation for dioxins/furans (Table 5-6)

5.4.3.3 Lead. Currently there are no toxicity values (i.e., CSFs or RfDs) available for the quantitative evaluation of potential human health impacts associated with exposures to lead in soils. According to the *Recommended Outline for Using Environmental Protection Agency Region IX Preliminary Remediation Goals in Screening Risk Assessments at Military Facilities* (Cal-EPA, 1994b), the PEA screening level of 130 ppm for lead in soils should be used. This

screening level for lead was used in conformance with Cal-EPA guidance (Cal-EPA 1994a) and as specified in the approved Workplan (Parsons ES, 1995).

5.4.3.4 Mercury. For evaluating the noncarcinogenic hazards associated with mercury, toxicity values for the inorganic form (mercuric chloride) were used. Toxicity values have been developed for the organic form (methyl mercury). However, there is no evidence to suggest that organic mercury is a COPC for Site FTIR-32A. Potential anthropogenic sources of metals at the site are believed to be associated with firing range activities, suggesting that inorganic forms of the metals predominate.

5.4.3.5 Polycyclic Aromatic Hydrocarbons. The USEPA has not yet established a national policy for assigning cancer potencies to different PAHs. In the interim, USEPA Region IX has set a regional policy based on the recommendation of the Environmental Criteria Assessment Office (ECAO) to use a set of toxicity equivalency factors (TEFs) to calculate a "benzo[a]pyrene equivalent" concentration for PAH mixtures. The toxicities associated with the various carcinogenic PAH COPCs detected in site soils were assigned using these potency equivalency factors (Table 5-9).

5.4.3.6 Total Petroleum Hydrocarbons. Currently there are no toxicity values (i.e., CSFs or RfDs) available for the quantitative evaluation of potential human health impacts associated with exposures to TPH. According to the *Recommended Outline for Using Environmental Protection Agency Region IX Preliminary Remediation Goals in Screening Risk Assessments at Military Facilities* (Cal-EPA, 1994b), TPH measurement should not be used at any level of risk assessment. This guidance states that the principle toxic constituents (i.e., benzene, toluene, ethylbenzene, xylenes, and PAHs) of hydrocarbon fuels should be evaluated. Where available, sampling results for these constituents were used in the quantitative evaluation of risks associated with non-specific petroleum hydrocarbons such as TRPH.

5.4.4 Toxicity Information Sources

The primary sources of toxicity values used in this baseline HHRA were the IRIS, 2000 compiled by USEPA, and the HEAST (USEPA, 1995). The Agency for Toxic Substance and Disease Registry (ATSDR) profiles for selected compounds were also reviewed. Toxicology profiles for the COPCs evaluated in this baseline HHRA are presented in Appendix C.

5.5 RISK CHARACTERIZATION

This section presents the methods and results of the risk characterization performed for the baseline HHRA. Risk characterization involves the integration of exposure estimates developed as part of the exposure assessment with dose-response information (toxicity values) developed as part of the toxicity assessment. The result is a quantitative estimate of the likelihood of chronic health effects, in the form of carcinogenic risks or noncarcinogenic hazards. The carcinogenic risk estimate is based on the premise that carcinogenicity is a non-threshold effect (i e , that even at the lowest dose there is some potential to develop carcinogenic effects). In contrast, the noncarcinogenic hazard estimate is based on the premise that for noncarcinogens there is a threshold dose below which adverse health effects will not occur.

5.5.1 Methods

Following are the methods that were used in the evaluation of carcinogenic risks and noncarcinogenic hazards for current/future military personnel and hypothetical future industrial workers potentially exposed to site-derived soil contaminants.

5.5.1.1 Carcinogenic Risks. Baseline human health risks were evaluated separately for carcinogenic effects and noncarcinogenic effects. The incremental lifetime cancer risk (ILCR) is an estimate of the increased risk of cancer due to lifetime exposure, at apportioned average daily doses, to constituents detected in each medium at the site. For current/future military personnel

and hypothetical future industrial workers, risks were calculated as the product of the exposure dose and the carcinogenic toxicity value, the CSF (USEPA, 1989).

The equation for calculating carcinogenic risks is as follows:

$$\text{ILCR (unitless)} = \text{CSF} \times \text{Dose}$$

where:

CSF = Cancer slope factor (mg/kg-day)-1
Dose = Exposure dose (mg/kg-day)

Cancer risks from multiple COPCs were assumed to be additive, and were summed to estimate a total cumulative ILCR for all carcinogenic site contaminants. The resulting risk estimates are an indication of the increased risk, above that applying to the general population, which may result from the exposures assumed for each scenario. The risk estimate is an upper bound estimate of risk, because of the protective assumptions used in the development of toxicity values and exposure estimates. Therefore, it is probable that the actual risks associated with potential exposures to site contaminants are lower than estimated risks.

5.5.1.2 Noncarcinogenic Hazards. To evaluate noncarcinogenic health effects due to potential exposures to site COPCs, a hazard quotient (HQ) was calculated for each COPC. The HQ was calculated as the ratio of the exposure dose to the RfD (USEPA, 1989).

The equation for calculating noncarcinogenic hazards is as follows:

$$HQ\ (unitless) = \frac{Dose}{RfD}$$

where:

Dose = Exposure dose (mg/kg-day)
RfD = Reference dose (mg/kg-day)

A hazard quotient greater than 1 0 indicates that the estimated exposure dose for that COPC may exceed acceptable health-protective levels for noncarcinogenic effects. Although an HQ of less than 1.0 suggests that noncarcinogenic health effects should not occur, an HQ of slightly greater than 1.0 is not necessarily an indication that adverse effects will occur.

The individual HQs for site COPCs were summed to produce a total cumulative hazard estimate, the HI. If the total HI estimate is less than 1.0, then no noncarcinogenic chronic health effects are expected to occur. If the total HI estimate is greater than 1.0, then adverse health risks are considered possible.

Sites that result in an estimated cumulative risk less than 1.0×10^{-6} and a cumulative HI less than 1.0 in the screening HHRA are generally recommended for no further action, in regard to human exposures. Sites for which the cumulative cancer risk is between 1.0×10^{-6} and 1.0×10^{-4} may be considered for no further action, depending upon site-specific considerations including current and potential future land uses. Sites that are associated with a cumulative cancer risk or noncancer HI greater than these criteria are generally considered for further action including potential evaluation of remedial alternatives (USEPA, 1991a).

5.5.2 Results

The results of the risk characterization performed for Site FTIR-32A are described in the following subsections. Detailed carcinogenic risk and noncarcinogenic hazard calculations for each media (i e., surface or subsurface soil) and receptor are presented in Appendix D. Summary results for Site FTIR-32A are presented in Table 5-10.

5.5.2.1 Surface Soils. The total cumulative cancer risk and noncancer hazard estimates for current/future military personnel exposed to surface soils were 3.0×10^{-6} and 2.4, respectively (Table 5-10). The cancer risk estimate is within USEPA's generally acceptable risk range of 1.0×10^{-6} to 1.0×10^{-4}, while the HI estimate slightly exceeds a hazard criterion of 1.0. The primary contributor to the cancer risk estimate for current/future military personnel was arsenic (ILCR = 3.0×10^{-6}). The primary contributor to excess hazard was manganese (HQ = 2.2). For no other COPCs were risk or HQ estimates in excess of 1.0×10^{-6} or 1.0, respectively. The exposure point concentration for arsenic in surface soils (6.3 mg/kg) was lower than the BUTL derived for arsenic at NTC Fort Irwin (9.14 mg/kg). Furthermore, all concentrations of arsenic detected in Site FTIR-32A surface soils were within the range of background for California soils (average = 3.5 mg/kg; range = 0.6 – 11) (Bradford et al., 1996). Similarly, the maximum concentration of manganese detected in Site FTIR-32A surface soils (649 mg/kg) was less than two-fold higher than the BUTL derived for manganese (361 mg/kg). In addition, all concentrations of manganese detected in Site FTIR-32A surface soils were within the range of background for California soils (average = 646 mg/kg; range = 253 – 1,687) (Bradford et al., 1996). Therefore, excess risk estimates for current/future military personnel are believed to be associated with naturally occurring ambient conditions.

The total cumulative cancer risk and noncancer hazard estimates for the hypothetical future industrial worker exposed to surface soils were 4.4×10^{-6} and 0.51, respectively (Table 5-10). These cancer risk and HI estimates are within USEPA's generally acceptable cancer risk range of 1.0×10^{-6} to 1.0×10^{-4}, and hazard criterion of 1.0. The primary contributor to the cancer risk estimate was arsenic (ILCR = 4.4×10^{-6}). As described above, however, arsenic concentrations in Site FTIR-32A surface soils, and the associated risks, are believed to represent naturally occurring ambient conditions.

5.5.2.2 Subsurface Soils. As described in Section 5.3.3, current/future military personnel are not anticipated to receive exposures to subsurface soils, and subsurface soil risks were not evaluated for this receptor. The total cumulative cancer risk and noncancer hazard estimates for the hypothetical future industrial worker exposed to subsurface soils were 7.3×10^{-6}

and 0 55, respectively (Table 5-10). These cancer risk and HI estimates are within USEPA's generally acceptable cancer risk range of 1.0×10^{-6} to 1.0×10^{-4}, and hazard criterion of 1.0. The maximum concentration of arsenic in subsurface soils (35.6 mg/kg) exceeded the BUTL derived for arsenic at NTC Fort Irwin (9.14 mg/kg). However, the exposure point concentration for arsenic in subsurface soils (9.9 mg/kg) only slightly exceeded the BUTL, and was within the typical range for California soils (average = 3 5 mg/kg; range = 0.6 – 11) (Bradford et al., 1996). No COPCs besides arsenic were associated with risk estimates in excess of 1.0×10^{-6}. Therefore, excess risk estimates for future industrial workers are within the range of those associated with naturally occurring ambient conditions.

5.6 UNCERTAINTY ANALYSIS

The presence of uncertainty is inherent in the risk assessment process. Generally, uncertainties in the risk assessment typically result from limitations in the available methods, information, and data used in the following:

- Characterization of contaminant sources
- Identification of site COPCs
- Evaluation of potential exposure scenarios and pathways
- Toxicity assessment
- Risk characterization

The uncertainties associated with each of these steps as they relate to the baseline HHRA for Sites FTIR-32A are described below.

5.6.1 Characterization of Contaminant Sources

There is a degree of uncertainty in the characterization of contaminant sources, since it is not possible to sample an entire site. The site investigations were based on site histories, known releases, and physical characteristics (e g , the presence of waste materials or topographic

anomalies). The nature of these site investigations focused on known or suspected sources of contamination. While it is believed that sufficient samples were collected to characterize the nature and extent of contamination at the sites, it is possible that areas not sampled may have also contained contaminants. However, sample locations were generally chosen such that they represented the area with the greatest potential to detect contaminants, if present

A total of 18 surface soil samples were collected from Site FTIR-32A and analyzed for inorganic constituents, VOCs, and TRPH. Surface soils samples were not analyzed for SVOCs or dioxins/furans. However, these constituents were detected in low concentrations, and with low detection frequencies (SVOCs), in subsurface soil samples. Only one subsurface soil sample was analyzed for dioxins/furans. However, this sampling location was selected to represent the highest probability of detecting dioxins/furans, and the associated risk estimate for dioxins/furans was low (2.1×10^{-15}). The soil sampling analytical results and the depths at which the subsurface samples were collected are presented in the Final Data Summary Report (Montgomery Watson, 1997).

5.6.2 Identification of Site Chemicals of Potential Concern

The process used in the selection of site COPCs may also introduce a degree of uncertainty in the baseline HHRA. However, protective assumptions were used in the selection of site COPCs. Chemicals selected for quantitative evaluation in the HHRA included all organic chemicals, and inorganic chemicals (other than essential nutrients) detected at concentrations above BUTLs established for Fort Irwin soils. For selenium and silver, BUTLs could not be established. To be protective, therefore, these chemicals were carried through the risk assessment as COPCs

5.6.3 Exposure Assessment

Because the exposure assessment is based on the estimation of potential rather than actual exposures, there is a degree of uncertainty in the dose estimate. The evaluation of industrial receptors under hypothetical future land use conditions was included in this baseline HHRA to

provide a basis for evaluating future land uses. However, Fort Irwin is not undergoing base closure, and the anticipated future land use for the range area is limited to military training exercises. Dose estimate calculations for the military worker were based on protective exposure assumptions. Furthermore, exposure point concentrations for all COPCs evaluated in this baseline HHRA were based on the maximum or 95 percent UCL concentration measured in soils.

5.6.4 Toxicity Assessment

There are also sources of uncertainty in the derivation of toxicity values (i.e., cancer slope factors and RfDs) used to quantify risks. Generally, the toxicity values that were used represent upper bound estimates, and incorporate uncertainty factors for extrapolation from animal data to humans, differences in individual sensitivity within populations, and the overall confidence in the dataset. Furthermore, the use of oral slope factors or oral RfDs for dermal toxicity values do not correct for differences in absorption and metabolism between the oral and dermal routes. Because the toxicity values established by USEPA are based on NOAEL concentrations and incorporate uncertainty factors, they are generally considered to be protective. The use of conservative toxicity values in the risk estimate tends to overestimate actual risks.

The risks associated with TPH were not evaluated quantitatively, because toxicity values are currently unavailable for these materials. However, analyses were performed for individual hydrocarbons which detect the most toxic constituents such as benzene, toluene ethylbenzene, and xylenes (BTEX) and PAHs. Toxicity values
are available for these constituents, and they were evaluated quantitatively in this baseline HHRA, when appropriate.

5.6.5 Risk Characterization

The different sources of uncertainty previously described are incorporated in the risk estimate. Because the majority of these uncertainties err on the conservative side, the risk estimate is considered to be protective. Furthermore, a 1.0 x 10^{-6} risk level does not equate to an actual

cancer incidence of one-in-one-million for substances that may cause cancer. The risk assessment process uses animal data to predict the probability of humans developing cancer over a 70-year lifetime. The estimated risks presented in this HHRA represent upper bound estimates; the actual risks are anticipated to be less.

6.0 ECOLOGICAL RISK EVALUATION

A screening-level predictive ERA was previously conducted for Sites FTIR-32A and FTIR-39 and the results were presented in the SI Report (Montgomery Watson, 1998). Briefly, the ecological assessment endpoints evaluated in the screening-level predictive ERA for Sites FTIR-32A and FTIR-39 included protection of the growth and survival of herbivorous consumer species and upper trophic level carnivorous species. The indicator receptors selected to represent consumer level and upper trophic level species were the Mojave ground squirrel (*Spermophilus mohavensis*) and golden eagle (*Aquila chrysaetos*), respectively. The measurement endpoints used to evaluate potential ecological impacts of soil contaminants identified at Site FTIR-32A and FTIR-39 were quantitative HI estimates for the Mojave ground squirrel and golden eagle.

For Site FTIR-32A, HI estimates for surface and subsurface soils exceeded 1.0 for the Mojave ground squirrel, but were less than 1.0 for the golden eagle. For Site FTIR-39, HI estimates for surface soil also exceeded 1.0 for the Mojave ground squirrel, and were less than 1.0 for the golden eagle. Potential ecological impacts associated with subsurface soils at Site FTIR-39 were not evaluated, due to the surficial nature of the contamination at this site (Montgomery Watson, 1998). Based on the results of the screening-level predictive ERA, Sites FTIR-32A and FTIR-39 were proposed for further evaluation to more accurately assess potential impacts to the Mojave ground squirrel (Montgomery Watson, 1998).

As part of the 1999 field investigation for NTC Fort Irwin, a biological field reconnaissance was conducted by representatives of the DTSC and CDFG on March 23, 1999 to evaluate the quantity and quality of habitat at several sites including Site FTIR-32A. As a result of the field reconnaissance, it was confirmed that Site FTIR-32A is mostly devoid of vegetation and provides inadequate habitat for the Mojave ground squirrel. Additionally, Site FTIR-39 was identified by Mickey Quillman (Natural and Cultural Resources Manager for NTC Fort Irwin) as also providing inadequate habitat for the Mojave ground squirrel (refer to Appendix B). Due to the absence of this receptor, or any other herbivorous consumer species, at Sites FTIR-32A and

FTIR-39, these sites were not further evaluated in this RI Report and are proposed for no further action in regard to ecological concerns.

7.0 CONCLUSIONS AND RECOMMENDATIONS

The following sections summarize the remedial investigation findings and the results of the screening and baseline risk assessments (BRAs) conducted for Sites FTIR-32A and FTIR-39. Sites where human risks or ecological hazards exceed generally accepted risk criterion in the BRA will be carried forward into a Feasibility Study, where remedial alternatives will be evaluated. Sites where human risks or ecological hazards do not exceed generally accepted risk criterion in the BRA will be proposed for no further action.

7.1 SITE FTIR-32A, LOWER GOAT MOUNTAIN LANDFILL

A burial pit containing metal debris and some burned material was identified at Site FTIR-32A Area 1. Only minor surface debris was identified in all other portions of Site FTIR-32 Soil samples collected in the vicinity of the burial pit contained detections of VOCs and SVOCs as well as elevated levels of metals

A screening HHRA was conducted as part of the Site Investigation for Site FTIR-32A (Montgomery Watson, 1998). Site FTIR-32A surface and subsurface soils were associated with exceedances of screening risk and hazard criteria for unrestricted (i.e., hypothetical future residential) land uses. Consistent with DTSC guidance (Cal-EPA, 1994b), Site FTIR-32A was proposed for further evaluation of potential human health impacts in a baseline risk assessment. Baseline human health risks were evaluated for current/future military personnel and hypothetical future industrial workers. The baseline cancer risk estimate for current/future military personnel exposed to surface soils was within USEPA's generally acceptable risk range of 1.0×10^{-6} to 1.0×10^{-4}, while the HI estimate slightly exceeded a hazard criterion of 1.0 The primary contributors to risk and hazard estimates for current/future military personnel were arsenic and manganese, which were judged to be representative of naturally occurring ambient conditions The baseline cancer risk and noncancer hazard estimates for hypothetical future industrial workers exposed to surface soils were within USEPA's generally acceptable cancer risk range and hazard criterion. Baseline risks for subsurface soil risks were only evaluated for hypothetical

future industrial workers, since current/future military personnel are not anticipated to receive exposures to subsurface soils (Section 5.3.3). Baseline cumulative cancer risk and noncancer hazard estimates for subsurface soils for the hypothetical future industrial worker were within USEPA's generally acceptable cancer risk range and hazard criterion. Baseline risks were also evaluated for a hypothetical future resident (i.e., unrestricted land use) based on 95% UCL concentrations. Cancer risk and noncancer hazard estimates for hypothetical future residents were within USEPA's acceptable risk range (refer to Table 7-1 and Appendix E). Therefore, Site FTIR-32A is proposed for no further action in regard to human health concerns.

The Final Site Inspection Report (Montgomery Watson, 1998) also included a screening-level predictive ERA for Site FTIR-32A. Screening-level ecological hazards were evaluated for the Mojave ground squirrel and golden eagle. The HI estimates for surface and subsurface soils exceeded 1.0 for the Mojave ground squirrel, but were less than 1.0 for the golden eagle. Based on the results of the screening-level predictive ERA, Site FTIR-32A was proposed for further ecological evaluation to more accurately assess potential impacts to the Mojave ground squirrel (Montgomery Watson, 1998). During a subsequent biological field reconnaissance conducted by representatives of DTSC and CDFG, however, it was concluded that Site FTIR-32A provides inadequate habitat for the Mojave ground squirrel. Therefore, Site FTIR-32A was not further evaluated in this RI Report in regard to potential ecological impacts, and is proposed for no further action in regard to ecological concerns.

In summary, Site FTIR-32A is proposed for no further action based on the continued operational use of this site as a range for firing and training maneuvers. In the event that this land use changes and/or NTC Fort Irwin enters into the base realignment and closure (BRAC) process, the baseline risk assessment for Site FTIR-32A should be re-evaluated in regard to human health concerns.

7.2 SITE FTIR-39, GOLDSTONE LAKE ROCKET TESTING RANGE

Several metals were detected in surface soil samples collected from Site FTIR-39 at concentrations above background levels. Explosives were not detected in any soil samples collected from the site. Although there were limited detections of petroleum hydrocarbons in surficial soils, it was not anticipated that petroleum hydrocarbons are present in Site FTIR-39 soils in concentrations likely to impact human health or the environment. Therefore, no additional site characterization activities were proposed for Site FTIR-39

A screening HHRA was conducted as part of the Site Investigation for Site FTIR-39 (Montgomery Watson, 1998). Screening risk and hazard estimates for Site FTIR-39 were below generally accepted risk and hazard criteria for unrestricted (i e , hypothetical future residential) and industrial land uses (Table 7-1). Therefore, Site FTIR-39 was proposed for no further evaluation in regard to human health concerns (Montgomery Watson, 1998).

The Final Site Inspection Report (Montgomery Watson, 1998) also included a screening-level predictive ERA for Site FTIR-39 The screening HI estimate exceeded 1.0 for the Mojave ground squirrel, but was less than 1.0 for the golden eagle. Based on the results of the screening-level predictive ERA, Site FTIR-39 was proposed for further ecological evaluation (Montgomery Watson, 1998). Subsequent to the screening ERA, Site FTIR-39 was identified by Mickey Quillman (Natural and Cultural Resources Manager for NTC Fort Irwin) as providing inadequate habitat for the Mojave ground squirrel (refer to Appendix B). During a visit to Site FTIR-39 DTSC and the Department of Fish & Game agreed that there is insufficient habitat at the site for the Mojave ground squirrel. Due to the absence of this receptor at Site FTIR-39, ecological impacts were not further evaluated in this RI Report and Site FTIR-39 is proposed for no further action in regard to ecological concerns.

In summary, Site FTIR-39 is proposed for no further action based on anticipated future land uses. Furthermore, the results of the screening HHRA suggest that Site FTIR-39 would be acceptable for unrestricted future land use in the event that the future status of NTC Fort Irwin is changed.

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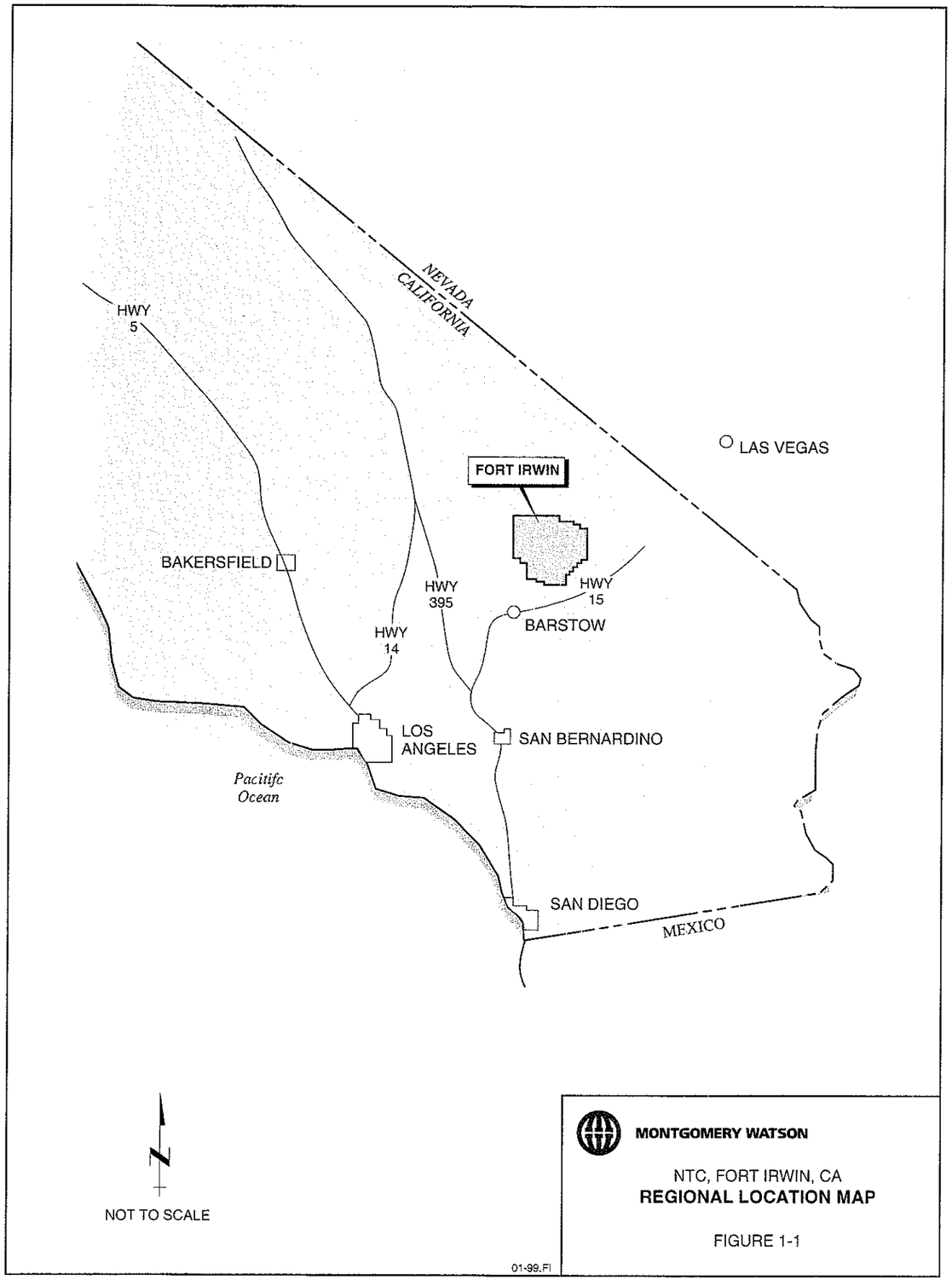

NEVADA
CALIFORNIA
HWY
5
LAS VEGAS
FORT IRWIN
BAKERSFIELD
HWY
395
HWY
15
BARSTOW
HWY
14
LOS
ANGELES
SAN BERNARDINO
Pacitifc
Ocean
SAN DIEGO
MEXICO
N
NOT TO SCALE
MONTGOMERY WATSON
NTC, FORT IRWIN, CA
REGIONAL LOCATION MAP
FIGURE 1-1
01-99.FI

CHINA LAKE
NAVAL WEAPONS
CENTER

AVAWATZ MOUNTAINS

Avawatz Valley

GRANITE MOUNTAINS

Drinkwater Dry Lake

FTIR-32A, Lower Goat
Mountain Landfill

Avawatz Lake

Red Pass Valley

Goldstone Dry Lake

East Range Road

Four Corners

FTIR-39, Goldstone Lake
Rocket Testing Range

Barstow Road

Goldstone Road

CANTONMENT
AREA

TIEFORT MOUNTAINS

Langford Lake Road

INSTALLATION
BOUNDARY

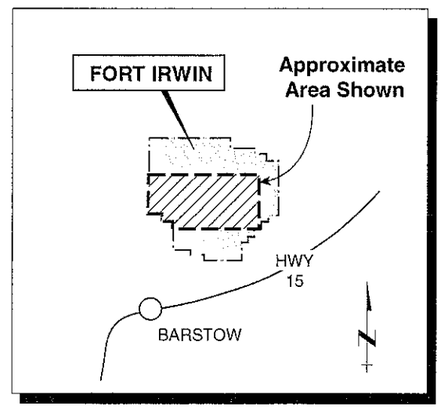
To
Barstow

Red Pass Dry Lake

LEGEND

- Roads
- Mountain Ranges
- Playas

0 2 4
SCALE IN MILES

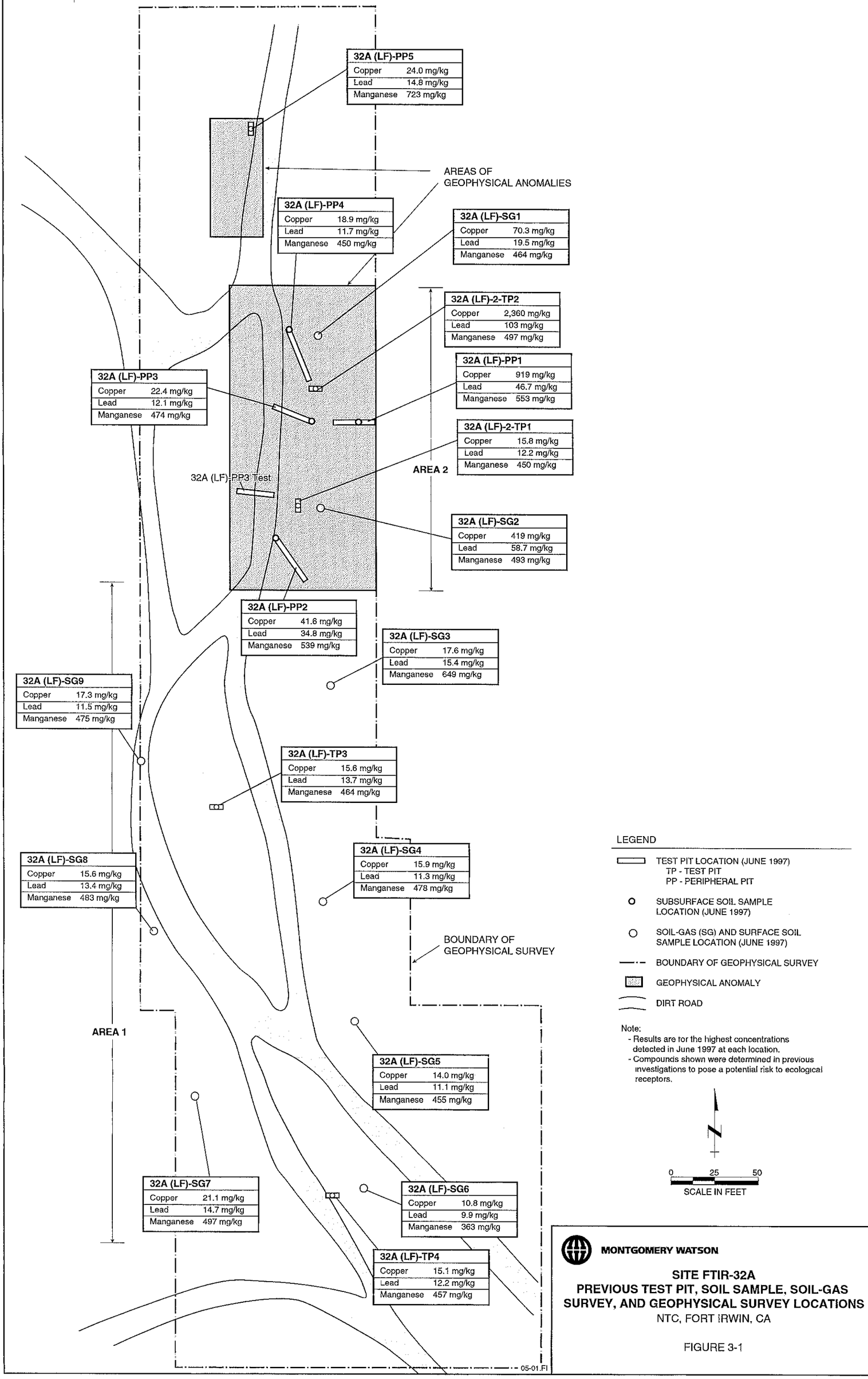


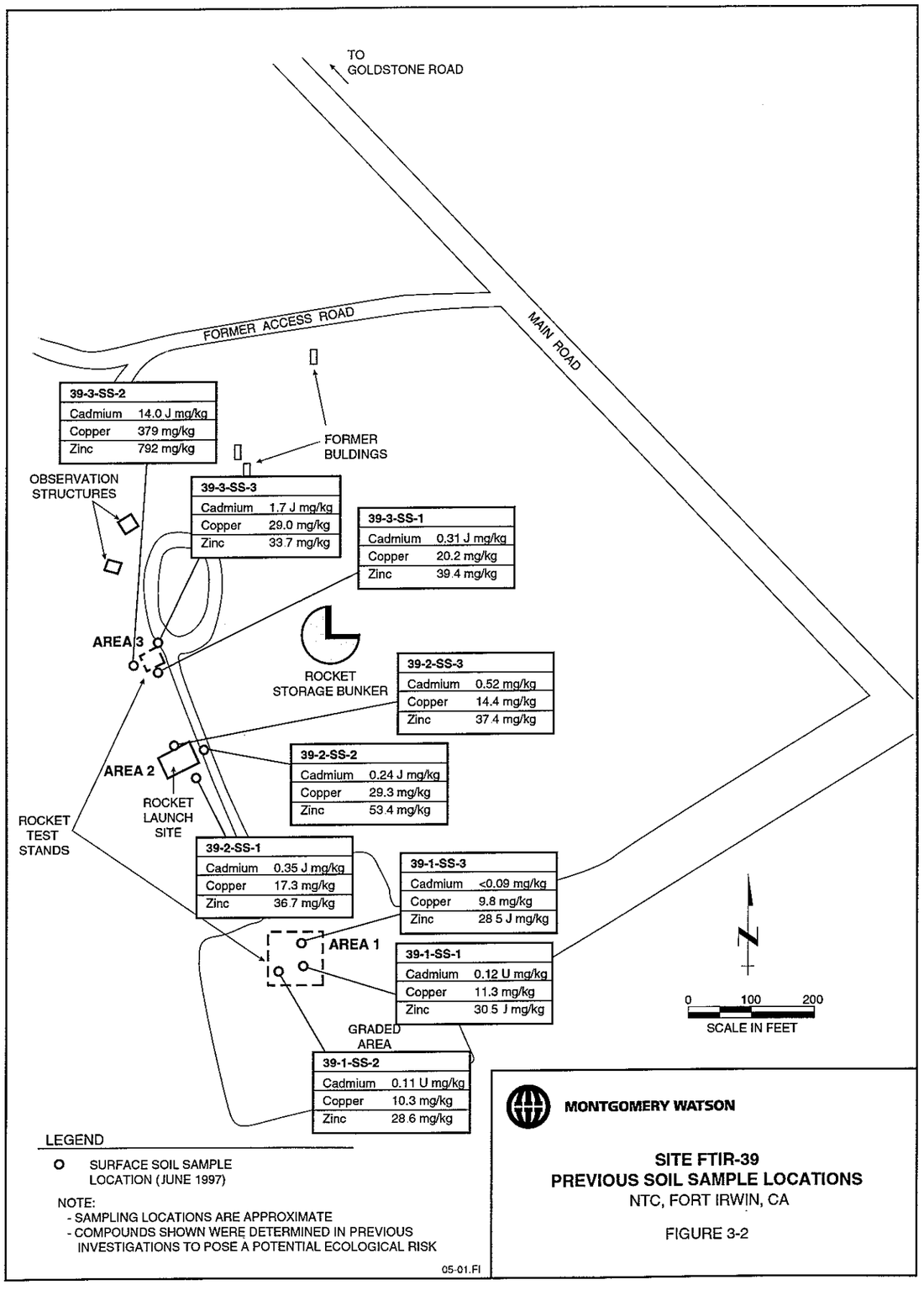
MONTGOMERY WATSON

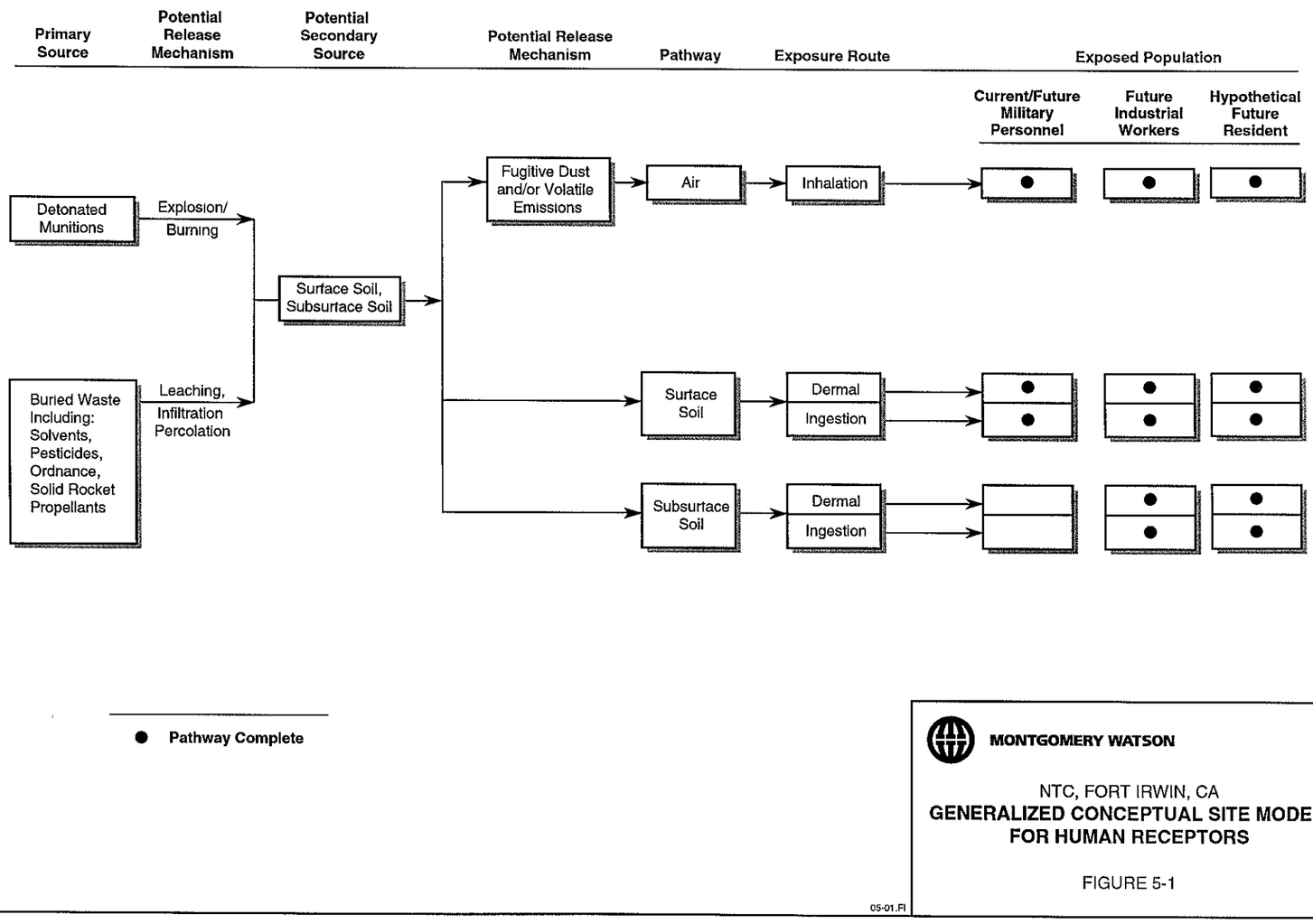
NTC, FORT IRWIN, CA
SITE LOCATION MAP

FIGURE 1-2

05-01.FI


32A (LF)-PP5
Copper 24.0 mg/kg
Lead 14.8 mg/kg
Manganese 723 mg/kg
AREAS OF GEOPHYSICAL ANOMALIES
32A (LF)-PP4
Copper 18.9 mg/kg
Lead 11.7 mg/kg
Manganese 450 mg/kg
32A (LF)-SG1
Copper 70.3 mg/kg
Lead 19.5 mg/kg
Manganese 464 mg/kg
32A (LF)-2-TP2
Copper 2,360 mg/kg
Lead 103 mg/kg
Manganese 497 mg/kg
32A (LF)-PP3
Copper 22.4 mg/kg
Lead 12.1 mg/kg
Manganese 474 mg/kg
32A (LF)-PP1
Copper 919 mg/kg
Lead 46.7 mg/kg
Manganese 553 mg/kg
32A (LF)-2-TP1
Copper 15.8 mg/kg
Lead 12.2 mg/kg
Manganese 450 mg/kg
32A (LF)-PP3 Test
AREA 2
32A (LF)-SG2
Copper 419 mg/kg
Lead 58.7 mg/kg
Manganese 493 mg/kg
32A (LF)-PP2
Copper 41.6 mg/kg
Lead 34.8 mg/kg
Manganese 539 mg/kg
32A (LF)-SG3
Copper 17.6 mg/kg
Lead 15.4 mg/kg
Manganese 649 mg/kg
32A (LF)-SG9
Copper 17.3 mg/kg
Lead 11.5 mg/kg
Manganese 475 mg/kg
32A (LF)-TP3
Copper 15.6 mg/kg
Lead 13.7 mg/kg
Manganese 464 mg/kg
32A (LF)-SG4
Copper 15.9 mg/kg
Lead 11.3 mg/kg
Manganese 478 mg/kg
32A (LF)-SG8
Copper 15.6 mg/kg
Lead 13.4 mg/kg
Manganese 483 mg/kg
BOUNDARY OF GEOPHYSICAL SURVEY
AREA 1
32A (LF)-SG5
Copper 14.0 mg/kg
Lead 11.1 mg/kg
Manganese 455 mg/kg
32A (LF)-SG7
Copper 21.1 mg/kg
Lead 14.7 mg/kg
Manganese 497 mg/kg
32A (LF)-SG6
Copper 10.8 mg/kg
Lead 9.9 mg/kg
Manganese 363 mg/kg
32A (LF)-TP4
Copper 15.1 mg/kg
Lead 12.2 mg/kg
Manganese 457 mg/kg
05-01.FI
LEGEND
TEST PIT LOCATION (JUNE 1997)
TP - TEST PIT
PP - PERIPHERAL PIT
SUBSURFACE SOIL SAMPLE LOCATION (JUNE 1997)
SOIL-GAS (SG) AND SURFACE SOIL SAMPLE LOCATION (JUNE 1997)
BOUNDARY OF GEOPHYSICAL SURVEY
GEOPHYSICAL ANOMALY
DIRT ROAD
Note:
- Results are for the highest concentrations detected in June 1997 at each location.
- Compounds shown were determined in previous investigations to pose a potential risk to ecological receptors.
N
0 25 50
SCALE IN FEET
MONTGOMERY WATSON
SITE FTIR-32A
PREVIOUS TEST PIT, SOIL SAMPLE, SOIL-GAS SURVEY, AND GEOPHYSICAL SURVEY LOCATIONS
NTC, FORT IRWIN, CA
FIGURE 3-1


TO
GOLDSTONE ROAD
FORMER ACCESS ROAD
MAIN ROAD
39-3-SS-2
Cadmium 14.0 J mg/kg
Copper 379 mg/kg
Zinc 792 mg/kg
FORMER
BULDINGS
OBSERVATION
STRUCTURES
39-3-SS-3
Cadmium 1.7 J mg/kg
Copper 29.0 mg/kg
Zinc 33.7 mg/kg
39-3-SS-1
Cadmium 0.31 J mg/kg
Copper 20.2 mg/kg
Zinc 39.4 mg/kg
AREA 3
ROCKET
STORAGE BUNKER
39-2-SS-3
Cadmium 0.52 mg/kg
Copper 14.4 mg/kg
Zinc 37.4 mg/kg
AREA 2
39-2-SS-2
Cadmium 0.24 J mg/kg
Copper 29.3 mg/kg
Zinc 53.4 mg/kg
ROCKET
LAUNCH
SITE
ROCKET
TEST
STANDS
39-2-SS-1
Cadmium 0.35 J mg/kg
Copper 17.3 mg/kg
Zinc 36.7 mg/kg
39-1-SS-3
Cadmium <0.09 mg/kg
Copper 9.8 mg/kg
Zinc 28 5 J mg/kg
AREA 1
39-1-SS-1
Cadmium 0.12 U mg/kg
Copper 11.3 mg/kg
Zinc 30 5 J mg/kg
N
0 100 200
SCALE IN FEET
GRADED
AREA
39-1-SS-2
Cadmium 0.11 U mg/kg
Copper 10.3 mg/kg
Zinc 28 6 mg/kg
LEGEND
SURFACE SOIL SAMPLE
LOCATION (JUNE 1997)
NOTE:
- SAMPLING LOCATIONS ARE APPROXIMATE
- COMPOUNDS SHOWN WERE DETERMINED IN PREVIOUS
INVESTIGATIONS TO POSE A POTENTIAL ECOLOGICAL RISK
05-01.FI
MONTGOMERY WATSON
SITE FTIR-39
PREVIOUS SOIL SAMPLE LOCATIONS
NTC, FORT IRWIN, CA
FIGURE 3-2



MONTGOMERY WATSON

NTC, FORT IRWIN, CA

GENERALIZED CONCEPTUAL SITE MODEL FOR HUMAN RECEPTORS

FIGURE 5-1

05-01.FI

TABLE 2-1

PLANTS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
Family Adiantaceae		
goldenback fern	*Pentagramma triangularis*	2
Family Cupressaceae		
Utah juniper	*Juniperus osteosperma*	2
Family Ephedraceae		
California tea	*Ephedra californica*	2, 4
Clokey tea	*Ephedra fasciculata v clokeyi*	2
Death Valley ephedra	*Ephedra funerea*	2, 3
Nevada tea	*Ephedra nevadensis*	2, 4
Green tea, squaw tea	*Ephedra viridis*	2, 4
DICOTS		
Family Amaranthaceae		
tumbling amaranth or pigweed	*Amaranthus albus*	2
spiny amaranth	*Amaranthus spinosus*	9
Arizona-sweet	*Tidestroemia oblongifolia*	2, 3
Family Apiaceae		
white flowered cymopterus	*Cymopterus aboriginum*	2, 3
veined cymopterus	*Cymopterus multinervatus*	11
Mojave wild parsley	*Lomatium mohavense*	2, 4
Parish's parsley or hog-fennel	*Lomatium nevadense v parishii*	1, 10
	Lomatium nevadense v pseudoorientale	3
Family Asclepiadaceae		
desert milkweed	*Asclepias erosa*	1, 2, 4
broom milkweed	*Asclepias subulata*	2, 4
Utah cynanchum	*Cynanchum utahense*	11
climbing milkweed	*Sarcostemma hirtellum*	1, 4, 11
Family Asteraceae		
goldenheads	*Acamptopappus sphaerocephalus*	1, 2, 4
dyssodia	*Adenophyllum (Dyssodia) cooperi*	1, 2, 4
burweed, sand-bur	*Ambrosia acanthicarpa*	2, 4
burrobush	*Ambrosia dumosa*	1, 2, 4
	Ambrosia dumosa X Hymenoclea salsola	
chaff-bush	*Amphipappus fremontii s fremontii*	1, 2, 4
scale-bud	*Anisocoma acaulis*	2
white western mugwort	*Artemisia ludoviciana s albula*	2, 4
bud-sage	*Artemisia spinescens*	1, 2, 4
Great Basin sagebrush	*Artemisia tridentata*	4
gravel-ghost	*Atrichoseris platyphylla*	2, 4
short-leaved broom baccharis	*Baccharis brachyphylla*	2
Emory baccharis	*Baccharis emoryi*	1, 2, 3, 4
mulefat	*Baccharis salicifolia*	1, 4
desert broom	*Baccharis sarathroides*	1
squaw waterweed	*Baccharis sergiloides*	1, 2, 4
lax flower	*Baileya pauciradiata*	2
desert marigold	*Baileya pleniradiata*	2, 4
sweetbush	*Bebbia juncea v aspera*	2, 4
spear-leaved brickellbush	*Brickellia arguta v arguta*	1, 2, 4
desert brickelbush	*Brickellia desertorum*	2
woolly brickelbush	*Brickellia incana*	2, 4
little-leaf brickelbush	*Brickellia microphylla*	10
pinyon brickelbush	*Brickellia oblongifolia v linifolia*	2

TABLE 2-1

PLANTS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
yellow tack-stem	*Calycoseris parryi*	2, 3, 4
pebble pincushion	*Chaenactis carphoclinia*	1, 2, 4
Fremont pincushion	*Chaenactis fremontii*	1, 2, 3, 4
Mojave pincushion	*Chaenactis macrantha*	2, 3, 4
desert pincushion	*Chaenactis stevioides*	1, 2
Xantu's pincushion	*Chaenactis xantiana*	1, 2
arid rubber rabbitbrush	*Chrysothamnus nauseosus s leiospermus*	1, 2
black-stem rabbitbrush	*Chrysothamnus paniculatus*	1, 4
round-leaf rabbitbrush	*Chrysothamnus teretefolius*	1, 2, 4
sticky-leaf rabbitbrush	*Chrysothamnus viscidiflorus s. puberulus*	2
Mojave thistle	*Circium mohavense*	1, 4
desert thistle	*Circium neomexicana*	1, 2, 4
Bigelow tickseed	*Coreopsis bigelovii*	1, 2, 4
bugseed	*Dicoria canescens*	1
brittlebush	*Encelia farinosa v. farinosa*	1, 2, 4
rayless encelia	*Encelia frutescens v. frutescens*	1, 2+, 4
Acton daisy	*Encelia actonii*	1, 2, 4
Virgin River encelia	*Encelia virginensis*	2+
Cooper's goldenbush	*Ericameria cooperi*	1, 2, 4
cliff goldenbush	*Ericameria cuneata v. spathulata*	1, 4
interior goldenbush	*Ericameria linearifolia*	1, 2
boulder fleabane	*Erigeron breweri v. porphyeticus*	1, 2, 10
tidy fleabane	*Erigeron pumilus v concinnoides*	1, 4
yellow frocks	*Eriophyllum ambiguum v. paleaceum*	1, 2, 3
Pringle woolly-sunflower	*Eriophyllum pringlei*	1, 2
Wallace's woolly-sunflower	*Eriophyllum wallacei*	1, 2, 4
Arizona filago	*Filago arizonica*	2
dwarf filago	*Filago depressa*	2
California gold, desert sunflower	*Geraea canescens*	1, 2, 4
keysia	*Glyptopleura marginata*	2
cudweed	*Gnaphalium palustre*	2
purple everlasting	*Gnaphalium purpureum*	9
snakeweed, small-flowered matchweed	*Gutierrezia microcephala*	1, 2, 4
annual sunflower	*Helianthus annuus*	2
cheesebush	*Hymenoclea salsola*	1, 4
alkali isocoma	*Isocoma acradenia v. eremophila*	1, 4
prickly lettuce	*Lactuca serriola*	1, 2, 4
autumn vinegarweed	*Lessingia lemmonii v. lemmonii*	2
Silver Lake aster	*Machaerantha arida*	1, 2, 3, 4
shrubby alkali aster	*Machaeranthera carnosa*	2
woolly dandelion	*Malacothrix californica*	1
snake's-head	*Malacothrix coulteri*	1, 2
desert dandelion	*Malacothrix glabrata*	1, 2, 4
Mojave desert stars	*Monoptilon bellidiforme*	2
desert stars	*Monoptilon bellioides*	1, 2, 4
hole-in-the-sand plant	*Nicolletia occidentalis*	1, 2, 4
Spanish needles	*Palafoxia arida v. arida*	1, 2, 4
cinch weed	*Pectis papposa*	1, 2
rock daisy	*Perityle emoryi*	1, 2, 3, 4
pygmy cedar	*Peucephyllum schottii*	1, 2, 4
arrowleaf	*Pleurocoronis pluriseta*	1, 2, 4

TABLE 2-1

PLANTS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
arrow-weed	*Pluchea (Tessaria) sericea*	1, 2, 4
odora	*Porophyllum gracile*	1, 2, 4
annual mitre	*Prenanthella exigua*	1, 2, 3
fan-leaf, turtlebacks	*Psathrotes annua*	1, 2, 4
velvet plant	*Psathrotes ramosissima*	1, 2, 4
California chicory	*Rafinesquia californica*	2
desert chicory	*Rafinesquea neomexicana*	1, 2
Mono grounsel	*Senecio flaccidus v. monoensis*	1, 2, 4
southern goldenrod	*Solidago confinis*	4
perennial sowthistle	*Sonchus arvensis*	4
sow-thistle	*Sonchus asper*	1, 2
common sow-thistle	*Sonchus oleraceus*	3, 4
annual milk-lettuce	*Stephanomeria exigua*	2, 4
rock pink	*Stephanomeria parryi*	2, 4
desert straw, wire-lettuce	*Stephanomeria pauciflora*	1, 2, 4
desert nest-straw	*Stylocline micropoides*	2
Peck nest-straw	*Stylocline psilocarphoides*	2
yellow xerasid, Fremont gold	*Syntrichopappus fremonti*	1, 2, 4
striped or silver cottonthorn	*Tetradymia argyraea**	2
cotton-thorn	*Tetradymia axillaris v axillaris*	1, 4
felt-thorn	*Tetradymia stenolepis*	2, 4
silver puffs	*Uropappus lindleyi*	2, 11
net-leaf daisy, leather leaf	*Viguiera reticulata*	1, 2
Mojave aster	*Xylorhiza tortifolia*	1, 2, 4
Family Bignoniaceae		
desert-willow	*Chilopsis linearis v arcuata*	1, 2, 4
Family Boraginaceae		
desert fiddleneck	*Amsinckia tessellata v tessellata*	1, 2, 4
narrow-leaved forget-me-not	*Cryptantha angustifolia*	2, 4
bearded forget-me-not	*Cryptantha barbigera*	2, 4
capped forget-me-not	*Cryptantha circumcissa*	2, 4
Clokey forget-me-not	*Cryptantha clokeyi**	2
golden cryptantha	*Cryptantha confertiflora*	2
wire-stem forget-me-not	*Cryptantha dumetorum*	2
prickly forget-me-not	*Cryptantha echinella*	2
rough-stemmed forget-me-not	*Cryptantha holoptera**	2
white-haired forget-me-not	*Cryptanta maritima*	2, 4
red-root cryptantha	*Cryptantha micrantha*	3, 4
pointed forget-me-not	*Cryptantha muricata*	1, 3, 6
Nevada forget-me-not	*Cryptantha nevadensis*	2, 4
wing-seed forget-me-not	*Cryptantha pterocarya*	1, 2, 4
mounded cryptantha	*Cryptantha tumulosa**	2
scented forget-me-not	*Cryptantha utahensis*	1, 2, 4
annual heliotrope	*Heliotropium convovulaceum v. californicum*	2
chinese pusley	*Heliotropium curvassavicum v. oculatum*	1, 2, 3, 4
chuckwalla comb-bur	*Pectocarya heterocarpa*	2
coastal comb-bur	*Pectocarya linearis v ferocula*	2
winged comb-bur	*Pectocarya penicillata*	2
broad-toothed comb bur	*Pectocarya platycarpa*	2
curve-fruited comb-bur	*Pectocarya recurvata*	2, 4
erect combbur	*Pectocarya setosa*	1, 2, 4

TABLE 2-1

PLANTS KNOWN TO OCCUR ON FORT IRWIN
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Common Name	Scientific Name	Source
Jones popcorn flower	*Plagiobothrys jonesii*	4
annual coldenia	*Tiquilia nuttallii*	1, 4
pleated coldenia	*Tiquilia plicata*	1, 2, 3, 4
Family Brassicaceae		
blue-podded rock-cress	*Arabis glaucovalvula*	1, 11
arched rock-cress	*Arabis perennans*	1, 2, 11
showy rock-cress	*Arabis pulchra v. gracilis*	1, 3
Sahara mustard	*Brassica tournefortii*	2
Cooper's caulanthus	*Caulanthus cooperi*	2, 3
desert candle	*Caulanthus inflatus*	2
yellow tansy mustard	*Descurainia pinnata*	2, 4
tansy-mustard	*Descurainia sophia*	2, 4
spectacle-pod	*Dithyrea californica*	2, 4
desert wallflower	*Erysimum capitatum*	2
desert crucifer	*Guillenia lasiophylla*	2, 4
yellow peppergrass	*Lepidium flavum v. flavum*	1, 2, 4
desert alyssum	*Lepidium fremontii*	1, 2, 4
hairy-pod peppergrass	*Lepidium lasiocarpum v. lasiocarpum*	2, 3, 4
cliff sibara	*Sibara rosulata*	1
tumble-mustard	*Sisymbrium altissimum*	2, 6
london rocket	*Sisymbrium irio*	2, 3
hedge-mustard	*Sisymbrium orientale*	2
prince's plume	*Stanleya pinnata v pinnata*	1, 2, 4
long-beaked twistflower	*Streptanthella longirostris*	2, 4
lace-pod	*Thysanocarpus curvipes*	2
fringe-pod	*Thysanocarpus laciniatus v. hitchockii*	1, 2, 4
Family Cactaceae		
cottontop cactus	*Echinocactus polycephalus v. polycephalus*	1, 2+, 4
hedgehog cactus	*Echinocereus engelmannii*	1, 2, 4
fishhook cactus	*Mamillaria tetrancistra*	2
beavertail	*Opuntia basilaris*	1, 4
silver or golden cholla	*Opuntia echinocarpa*	1, 4
Mojave prickly pear	*Opuntia erinacea*	1
pencil cholla	*Opuntia ramosissima*	1, 2, 4
Family Campanulaceae		
sticky threadstem	*Nemacladus glanduliferus v orientalis*	2, 4
yellow-flowered threadstem	*Nemocladus rubescens*	2
Inyo threadstem	*Nemacladus sigmoideus*	2
parishella	*Parishella californica*	2
Family Capparidaceae		
Mojave stinkweed	*Cleomella obtusifolia*	1, 2, 3, 4
Family Caprifoliaceae		
fragrant snowberry	*Symphoricarpus longiflorus*	1, 3, 4
Family Caryophyllaceae		
Achyronychia cooperi	*frost-mat*	2, 3, 6
desert sandwort	*Arenaria macradenia v macradenia*	1, 2, 4
saltmarsh sand-spurry	*Spergularia marina*	1
Family Chenopodiaceae		
iodine brush	*Allenrolfea occidentalis*	2, 4
four-wing saltbush	*Atriplex canescens*	1, 2, 4
shadscale	*Atriplex confertifolia*	1, 2, 4

TABLE 2-1

PLANTS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
wheelscale	*Atriplex elegans v elegans*	2, 4
wheelscale	*Atriplex elegans v fasciculata*	2, 3
desert holly	*Atriplex hymenelytra*	1, 2, 4
Nevada saltbush	*Atriplex lentiformis s. torreyi*	1, 2, 4
Parry saltbush	*Atriplex parryi*	1, 2, 4
arrowscale	*Atriplex phyllostegia*	2, 3, 4
allscale	*Atriplex polycarpa*	1, 2, 3, 4
spinescale	*Atriplex spinifera*	2
5-hook bassia	*Bassia hyssopifolia*	5
hoary goosefoot	*Chenopodium incanum*	2
lambsquarter, garden goosefoot	*Chenopodium murale*	1, 3
spiny hop-sage	*Grayia spinosa*	1, 2, 4
summer-cypress	*Kochia americana*	9
winter fat	*Krascheninnikovia lanata*	1, 2+, 4
patata	*Monolepis nuttalliana*	1, 2, 4
alkali-pink	*Nitrophila occidentalis*	2
barb-wire tumbleweed	*Salsola paulsenii*	2, 3, 4
tumbleweed, Russian thistle	*Salsola tragus (australis)*	1, 2, 4
greasewood	*Sarcobatus vermiculatus*	9
bush seepweed, inkweed	*Suaeda moquinii*	1, 2, 4
Family Convovulaceae		
alkali weed	*Cressa truxillensis v. vallicola*	1, 2, 3
dodder	*Cuscuta californica*	1, 2, 4
toothed dodder	*Cuscuta denticulata*	2
Family Cucurbitaceae		
coyote melon	*Cucurbita palmata*	1, 2, 4
Family Euphorbiaceae		
rattlesnake weed	*Chamaesyce albomarginata*	2, 4
desert annual spurge	*Chamaesyce micromeria*	1, 2
valley spurge	*Chamaesyce ocellata*	1, 2
Parish's spurge	*Chamaesyce parishii*	2
perennial spurge	*Chamaesyce polycarpa*	1, 2
Yuma spurge	*Chamaesyce setiloba*	3
Death Valley spurge	*Chamaesyce vallis-mortae*	1, 4
California croton	*Croton californica*	1, 2, 3, 4
turkey mullein, doveweed	*Eremocarpus setigerus*	1
toothleaf	*Stillingia paucidentata*	2, 4
spiny stillingia	*Stillingia spinulosa*	1, 2, 3, 4
Family Fabaceae		
keel beak	*Astragalus acutirostris*	2, 3
dwarf locoweed	*Astragalus didymocarpus didymocarpus*	2
2-seeded locoweed	*Astragalus didymocarpus v. dispermus*	4
Lane Mountain milkvetch	*Astragalus jaegerianus**	2+, 4
Catherine's milkvetch	*Astragalus layneae*	2, 4
Fremont rattleweed	*Astragalus lentiginosus v. fremontii*	2, 4
common rattleweed, dapplepod	*Astragalus lentiginosus v. variabilis*	2, 4
Mojave milkvetch	*Astragalus mohavensis v. hemigyrus*	3
Pursh's milkvetch	*Astragalus purshii v. tinctus*	1, 2, 3, 4
downy dalea	*Dalea mollisima*	1, 2, 4
short-podded lotus	*Lotus humistratus*	2, 4
Mojave rock-pea	*Lotus rigidus*	1, 2, 4

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PLANTS KNOWN TO OCCUR ON FORT IRWIN
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Common Name	Scientific Name	Source
coast lotus	*Lotus salsuginosus v. brevivexillus*	4
stiff-haired lotus	*Lotus strigosus (tomentellus)*	1, 2, 4
Chilean lotus	*Lotus wrangelianus (subpinnatus)*	2+
Arizona lupine	*Lupinus arizonicus*	1
bajada lupine	*Lupinus concinnus v. orcuttiana*	2, 4
yellow-eyes	*Lupinus flavoculatus*	2
sunrise lupine	*Lupinus microcarpus v. horizontalis*	2, 4
royal lupine	*Lupinus odoratus*	2, 4
pale lupine	*Lupinus pallidus*	1
sand lupine	*Lupinus shockleyi*	2, 3, 4
honey mesquite	*Prosopis glandulosa v. torreyana*	1, 2, 3, 4
screwbean mesquite	*Prosopis pubescens*	1, 2, 3, 4
Mojave indigo bush	*Psorothamus arborescens v. arborescens**	2, 3, 4
Indigo bush	*Psorothamnus fremontii v. attenuata*	1, 4, 12
spotted dalea	*Psorothamnus polydenius v polydenius*	1, 2, 4
desert senna	*Senna (Cassia) armata*	1, 2, 4
pinpoint clover	*Trifolium gracilentum v. gracentum*	2
Family Frankeniaceae		
alkali heath	*Frankenia salina (grandifolia v campestris)*	1, 3
Family Geraniaceae		
red-stemmed filaree	*Erodium cicutarium*	1, 2+, 4
Texas filaree	*Erodium texanum*	1, 2, 4
Family Hydrophyllaceae		
whispering bells	*Emmenanthe penduliflora*	2, 3, 4
spotted eucrypta	*Eucrypta chrysanthemifolia v. bipinnatifida*	2, 3, 4
desert eucrypta	*Eucrypta micrantha*	2
purple mat	*Nama demissum v. covillei*	2
purple mat	*Nama demissum demissum*	2, 4
narrow-leaved nama	*Nama depressum*	2
hairy nama	*Nama hispidium v spathulatum*	1, 2
small-leaved nama	*Nama pusillum*	2
wild heliotrope	*Phacelia crenulata v. ambigua*	2
Notch-leaf phacelia	*Phacelia crenulata v. crenulata*	4
limestone phacelia	*Phacelia cryptantha*	2, 3
blue-flowered heliotrope	*Phacelia distans*	2, 4
yellow throats	*Phacelia fremontii*	2, 4
weasel phacelia	*Phacelia mustelina*	2?
alkali phacelia	*Phacelia neglecta*	2
black tack phacelia	*Phacelia pachyphylla*	2, 3, 4
specter phacelia	*Phacelia pedicellata*	2, 4
round-leaved phacelia	*Phacelia rotundifolia*	2, 3, 4
California hyacinth	*Phacelia tanacetifolia*	1, 2, 4
Death Valley phacelia	*Phacelia vallis-mortae*	2, 4
fiesta flower	*Pholistoma membranaceum*	2, 4
Family Krameriaceae		
Pima ratany	*Krameria erecta (parvifolium v. imparata)*	1, 2, 4
Family Lamiaceae		
horehound	*Marrubium vulgare*	1, 4
spearmint	*Mentha spicata*	9
horsemint pennyroyal	*Monardella linoides s. linoides*	2
bladder sage	*Salazaria mexicana*	1, 2, 4

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PLANTS KNOWN TO OCCUR ON FORT IRWIN
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Common Name	Scientific Name	Source
thistle sage	*Salvia carduacea*	1, 2, 4
chia sage	*Salvia columbariae*	1, 2, 4
Mojave sage	*Salvia mohavensis*	1, 2, 3, 4
showy sage	*Salvia pachyphylla*	2, 4
Family Lennoaceae		
scaly sandfood	*Pholistoma arenarium*	2, 4
Family Loasaceae		
rock-nettle	*Eucnide urens*	1, 2, 4
blazing-star	*Mentzelia affinis*	2
white-stem blazing star	*Mentzelia albicaulis*	2, 4
desert blazing star	*Mentzelia desertorum*	2
sand blazing star	*Mentzelia involucrata v. involucrata*	2, 4
obscure blazing star	*Mentzelia obscura*	2, 3
Inyo blazing star	*Menzelia oreophila*	2
wing-seeded blazing star	*Mentzelia pterosperma*	3
shiny-leaf sandpaper plant	*Petalonyx nitidus*	2
sandpaper plant	*Petalonyx thurberi s. thurberi*	1, 4
Family Lythraceae		
California loosestrife	*Lythrum californicum*	2
Family Malvaceae		
white mat mallow	*Eremalche exilis*	2, 4
desert five-spot	*Eremalche rotundifolia*	1, 2, 4
apricot mallow	*Sphaeralcea ambigua v. ambigua*	1, 2, 4
Family Nyctaginaceae		
desert sand-verbena	*Abronia villosa v villosa*	1, 2, 4
windmills	*Allionia incarnata*	2, 4
ringstem	*Anulocaulis annulata*	2
wishbone plant	*Mirabilis bigelovii v. bigelovii*	2, 4
wishbone plant	*Mirabilis bigelovii v retrorsa*	1, 2
Family Oleaceae		
desert olive	*Foresteria pubescens (neomexicana)*	1, 8
Family Onagraceae		
dwarf woody bottlewasher	*Camissonia boothii v condensata*	2, 4
desert woody bottlewasher	*Camissonia boothii v desertorum*	2, 3, 4
desert suncups	*Camissonia brevipes v. brevipes*	1, 4
field-primrose	*Camissonia campestris s. campestris*	1, 2, 4
brown-eyed evening primrose	*Camissonia claviformis v. claviformis*	1, 2, 3, 4
yellow flowered brown-eyed evening primrose	*Camissonia claviformis v. piersonii*	4
gilman suncups	*Camissonia kernensis s. gilmanii*	2
narrow-leaved evening-primrose	*Camissonia refracta*	2, 4
wavy-leaved evening-primrose	*Oenothera caespitosa s. crinita*	2
bird cage primrose, devils lantern	*Oenothera deltoides s deltoides*	1, 2, 3
yellow primrose	*Oenothera primaveris s. primaveris*	2, 4
Family Orobanchaceae		
	Orobanche sp	4
Family Papaveraceae		
prickle poppy	*Argemone corymbosa*	2, 3, 4
Mojave gold poppy	*Eschscholzia glyptosperma*	1, 2, 3, 4
small-flowered gold poppy	*Eschscholzia minutiflora*	1, 2, 3, 4
Family Plantaginaceae		
woolly plantain	*Plantago ovata (insularis v. fastigiata)*	1, 2, 4

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PLANTS KNOWN TO OCCUR ON FORT IRWIN
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Common Name	Scientific Name	Source
Patagonia plantain	*Plantago patagonica v. patagonica*	4
Family Polemoniaceae		
Mojave wooly star-flower	*Eriastrum densiflorum v. mohavensis*	1
blue mantle	*Eriastrum diffusum*	2
sapphire flower	*Eriastrum eremicum*	1, 4
southern gilia	*Gilia australis*	3, 11
inyokern gilia	*Gilia brecciarum v neglecta*	2, 4, 10
showy gilia	*Gilia cana v. speciformis*	1, 2, 4
Clokey's gilia	*Gilia clokeyi*	4
thread-stemmed gilia	*Gilia filiformis*	1, 2, 3, 4
broad-leaved gilia	*Gilia latifolia*	4
Great Basin gilia	*Gilia leptomeria*	2
dainty gilia	*Gilia micromeria*	2, 3
dwarf gilia	*Gilia minor*	2, 4
volcanic pale gilia	*Gilia ochroleuca v ochroleuca*	4
star gilia	*Gilia stellata*	1, 2, 3, 4
desert gilia	*Gilia transmontana*	1, 4, 10
spreading ipomopsis	*Ipomopsis polycladon*	1
desert calico	*Loeselliastrum matthewsii*	2, 3, 4
sunbonnets	*Loeselliastrum schottii*	2
dotted sunbonnets	*Langloisia setosissima s. punctata*	1, 2, 4
bristly sunbonnets	*Langloisia setosissima s. setosissima*	4
prickly phlox	*Leptodactylon pungens v. pungens*	2, 4
desert linanthus	*Linanthus demissus*	2, 4
evening snow	*Linanthus dichotomus*	2, 3, 4
jones linanthus	*Linanthus jonesii*	1, 2
Family Polygonaceae		
red triangles	*Centrostegia thurberi*	2
brittle spineflower	*Chorizanthe brevicornu*	1, 2, 4
badlands spineflower	*Chorizanthe corrugata*	1, 3
rigid spiny herb	*Chorizanthe rigida*	1, 2+, 4
Watson's spineflower	*Chorizanthe watsonii*	2
white skeletonweed	*Eriogonum baileyi*	2, 4
short-flowered skeletonweed	*Eriogonum brachyanthum*	2
tecopa skeletonweed	*Eriogonum brachypodum*	2
flat-topped skeletonweed	*Eriogonum deflexum v. deflexum*	2
Desert bush buckwheat	*Eriogonum fasciculatum v polifolium*	1, 2, 4
	Eriogonum gracile	3
slender skeletonweed	*Eriogonum gracillimum*	1, 4
woolly heerman's buckwheat	*Eriogonum heermannii v floccosum**	2, 4
desert trumpet	*Eriogonum inflatum*	1, 2, 4
spotted skeletonweed	*Eriogonum maculatum*	2
Great Basin Buckwheat	*Eriogonum cf. Microthecum v. ambiguum*	4, 6
bird's-nest skeletonweed	*Eriogonum nidularium*	1, 2, 4
palmers bird cage	*Eriogonum plamerianum*	2
turk's cap	*Eriogonum pusillum*	1, 2, 4
kidney-leaf skeletonweed	*Eriogonum reniforme*	1, 2, 4
thomas skeletonweed	*Eriogonum thomasii*	2, 4
little trumpets	*Eriogonum trichopes v trichopes*	1, 2, 4
sulfur buckwheat	*Eriogonum umbellatum v. ferrissii*	9
sulfur buckwheat	*Eriogonum umbellatum v. versicolor*	9

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PLANTS KNOWN TO OCCUR ON FORT IRWIN
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Common Name	Scientific Name	Source
Wright's buckwheat	*Eriogonum wrightii*	2+
punctured bract	*Mucronea perfoliata*	2+
red saucers	*Oxytheca perfoliata*	1, 2, 4
knotweed	*Polygonum argyrocoleon*	2
wild rhubarb	*Rumex hymenosepalus*	1, 4
purple dock	*Rumex violascens*	2
Family Portulaceae		
pussypaws	*Calyptridium monandrum*	1
miner's lettuce	*Claytonia parviflora*	2, 3
Family Ranunculaceae		
desert larkspur	*Delphinium parishii*	1, 2, 4
Family Resedaceae		
oligomeris	*Oligomeris linifolius*	1, 2, 3, 4
Family Rhamnaceae		
Gray thorn	*Zizyphus cf. Obtusifolia v. canescens*	8
Family Rosaceae		
blackbrush	*Coleogyne ramosissima*	1, 2, 4
desert almond	*Prunus fasciculata*	2+, 4
bitterbrush	*Purshia mexicana v. stansburyana*	8
Family Rubiaceae		
star bedstraw	*Galium stellatum s. eremicum*	1, 2, 4
Family Rutaceae		
turpentine-broom	*Thamnosma montana*	1, 2, 4
Family Salicaceae		
Fremont cottonwood	*Populus fremontii*	1, 2, 3, 4
Narrow-leaved willow	*Salix exigua*	2
Goodding's black willow	*Salix gooddingii*	1, 2
Family Saururaceae		
yerba mansa	*Anemopsis californica*	1, 2, 3, 4
Family Scrophulariaceae		
twining snapdragon	*Antirrhinum filipes*	4
desert Indian paintbrush	*Castilleja angustifolia*	2, 3
purple owl's clover	*Castilleja exerta*	2
toad-flax paintbrush	*Castilleja linariifolia*	4
desert keckiella	*Keckiella antirrhinoides s. microphylla*	4
wash monkeyflower	*Mimulus bigelovii v. bigelovii*	1, 2, 4
common monkeyflower	*Mimulus guttatus*	2
lesser mohavea	*Mohavea breviflora*	2, 3
beardtongue	*Penstemon sp*	2
Palmer's beardtongue	*Penstemon palmeri*	2
brookline	*Veronica cf. Anagallis-aquatica*	2+
Family Solanaceae		
Jimson weed	*Datura wrightii*	2
wolfberry	*Lycium andersonii*	1, 2, 4
peach-thorn	*Lycium cooperi*	1, 2, 4
rabbit-thorn	*Lycium pallidum v. oligospermum*	2
desert tobacco	*Nicotiana obtusifolia (trigonophylla)*	1, 2, 4
ground cherry	*Physalis crassifolia*	1, 2, 4
husk cherry	*Physalis hederifolia v. fendleri*	9
gray husk cherry	*Physalis pubescens v. grisea (pruinosa)*	4
white flowered nightshade	*Solanum americanum*	2

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PLANTS KNOWN TO OCCUR ON FORT IRWIN
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Common Name	Scientific Name	Source
Family Tamaricaceae		
four-petaled salt cedar	*Tamarix parviflora*	2, 3
salt cedar	*Tamarix ramosissima*	1, 2, 4
Family Viscaceae		
desert mistletoe	*Phoradendron californicum*	1, 2, 4
Family Zygophyllaceae		
creosote bush	*Larrea tridentata*	1, 2, 4, 11
puncture vine	*Tribulus terrestris*	2
MONOCOTS		
Family Agavaceae		
Joshua tree	*Yucca brevifolia v. brevifolia*	1, 2+, 4
Mojave yucca	*Yucca schidigera*	1, 2+, 4
Family Cyperaceae		
spike rush	*Eleocharis macrostachya*	9
Mexican spike rush	*Eleocharis montevidensis*	2
Parish's spike rush	*Eleocharis parishii*	6, 9
hard stem bullrush	*Scirpus acutus*	2
bullrush	*Scirpus (olneyi) americanus*	2, 4
tri-square	*Scirpus pungens*	2
alkali bullrush	*Scirpus robustus*	4
Family Juncaceae		
wire grass	*Juncus balticus*	1, 3, 4
toad rush	*Juncus bufonius*	1, 3
Cooper's rush	*Juncus cooperi*	1, 3, 4
wire grass	*Juncus mexicanus*	1, 2
Family Liliaceae		
crested onion	*Allium atrorubens v. cristatum*	1, 3, 4
Kennedy mariposa lily	*Calochortus kennedyi*	2
alkali mariposa lily	*Calochortus striatus**	2
blue dycks	*Dichelostemma capitatum*	2, 4
desert lily	*Hesperocallis undulata*	3, 4
crowned onion	*Muilla coronata**	4
Family Poaceae		
Indian rice grass	*Achnatherum (Oryzopsis) hymenoides*	1, 2, 4
desert needle grass	*Achnatherum (Stipa) speciosa*	2, 4
six-week threeawn	*Aristida adscensionis*	1, 4
California threeawn	*Aristida californica*	2
purple three-awn	*Aristida purpurea v. nealleyi (A glauca)*	2, 4
eyelash grass	*Bouteloua barbata*	2
ripgut brome	*Bromus diandrus*	2
red brome	*Bromus madritensis s. rubens*	1, 2+, 4
cheatgrass	*Bromus tectorum*	2, 4
Chilean chess	*Bromus trinii*	1, 2, 4
finger grass	*Chloris virgata*	9
Bermuda grass	*Cynodon dactylon*	9
saltgrass	*Distchlis spicata*	1, 2, 3, 4
barnyard grass	*Echinochloa crus-galli*	9
squirrel-tail grass	*Elymus elymoides (Sitanion hystrix)*	2, 4
fluff grass	*Erioneuron pulchellum*	1, 4
foxtail barley	*Hordeum murinum s. leporinum*	1, 2, 3, 4

TABLE 2-1

PLANTS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
Mexican sprangletop	*Leptochloa uninervia*	9
salina wildrye	*Leymus salinus*	2
annual ryegrass	*Lolium perenne*	2+
tall melic grass	*Melica frutescens*	1, 2, 4
deergrass	*Muhlenbergia rigens*	2, 4
canary grass	*Phalaris caroliniana*	2
common reed	*Phragmites australis*	1, 4
big galleta grass	*Pleuraphis (Hilaria) rigida*	1, 2, 4
rush-leaved blue grass	*Poa secunda* s. *juncifolia*	2, 3
Nevada blue grass	*Poa secunda* s. *secunda*	2, 4
rabbits foot grass	*Polypogon monspeliensis*	1, 2, 3, 4
Arabian grass	*Schismus arabicus*	2, 4
split grass	*Schismus barbatus*	2, 4, 6
alkali dropseed	*Sporobolus airoides*	2, 4
fescue grass	*Vulpia microstachys* v. *microstachys*	2
fescue grass	*Vulpia microstachys* v. *pauciflora*	2
six-weeks fescue	*Vulpia octoflora*	2, 4
Family Typhaceae		
southern cattail	*Typha domingensis*	2
broad-leaved cattail	*Typha latifolia*	4

Notes:
* - Sensitive Species occurring on Fort Irwin
s. - subspecies
v. - variety
cf - identification in doubt, based on sterile specimen or no voucher in existence.
2+ - no vouchers made but based on positive identification.

TABLE 2-1

PLANTS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Sources:

1 Plant list compiled by Army Corps botanist Lois Goodman Plant species list, Fort Irwin National Training Center, San Bernardino County, California.

2 Gibson, A., B Prigge, and K. Niessen. Floral Surveys at the Fort Irwin Training Center, San Bernardino County, California. Includes vouchered specimens collections made at NTC and deposited at Rancho Santa Ana Botanic Gardens and U.C.L.A herbaria.

3 1970s Hendrickson, James. Field collections at Rancho Santa Ana Botanic Gardens and documented in Gibson et al.

4 Lee and Ro Consulting Engineers compiled by Mark Bagley field work conducted with John Chestnut and Denise Labrouteux Endangered and sensitive species survey and deficiency tabulation for Fort Irwin National Training Center and Goldstone Space Communications Complex. Prepared for Directorate of Engineering and Housing, Fort Irwin National Training Center.

5 David Charlton field notes. Floral survey of Garlic Springs, 1991.

6 AEHA observed 125 species at study plots.

7 Brand, C ; W. Rickard, and N Cadoret Vegetations Studies National Training Center, Fort Irwin, California

8 Pratt, Gordon. Field work for 1995 butterfly surveys at Ft. Irwin Per Comm between Steve Ahmann and David Charlton, 1996.

9 LCTA, 1991-1994. Conducted by Colorado State University students

10 Jaeger Edmund. Field collections stored at Rancho Santa Ana Botanic Gardens. Biology instructor Riverside City College.

11 Wolfe, Carl, and Philip Munz. Field collections by the horticulturalist and taxonomist at Rancho Santa Ana Botanic Gardens.

12 De Decker, Mary. Field collections housed at her herbarium at Independence with duplicates at Rancho Santa Ana Botanic Gardens.

TABLE 2-2

REPTILES KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
CLASS REPTILIA		
Testudinidae		
desert tortoise	*Gopherus agassizii**	1
Gekkonidae		
western banded gecko	*Coleonyx variegatus*	1, 4
Iguanidae		
desert iguana	*Dipsosaurus dorsalis*	1, 4
chuckwalla	*Sauromalus obesus**	1,4
Mojave fringe-toed lizard	*Uma scoparia**	11
zebra-tailed lizard	*Callisaurus draconoides*	1, 4
desert collared lizard	*Crotaphytus insularis*	1, 4
long-nosed leopard lizard	*Gambilia wislizenii*	1, 4
desert spiny lizard	*Sceloporus magister*	1, 4
western fence lizard	*Sceloporus occidentalis*	11
longed-tailed brush lizard	*Urosaurus graciosus*	11
side-blotched lizard	*Uta stansburiana*	1, 4
desert horned lizard	*Phrynosoma platyrhinos*	1, 4
Xantusidae		
desert night lizard	*Xantusia vigilis*	1, 4
Teiidae		
western whiptail	*Cnemidophorus tigris*	1, 4
Scincidae		
Gilbert skink	*Eumeces gilberti*	4
Leptotyphlopidae		
western blind snake	*Leptotyphlops humilis*	11
Boidae		
rosy boa	*Lichanura trivirgata*	1
Colubridae		
spotted leaf-nosed snake	*Phyllorhynchus decurtatus*	1, 4
coachwhip	*Masticophis flagellum*	1, 4
western patch-nosed snake	*Salvadora hexalepis*	1, 4
glossy snake	*Arizona elagans*	1, 4
gopher snake	*Pituaphis melanoleucus*	1, 4
common kingsnake	*Lampropeltis getulus*	1, 4
long-nosed snake	*Rhinocheilus lecontei*	1, 4
ground snake	*Sonora semiannulata*	11
western shovel-nosed snake	*Chionactis occipitalis*	1, 4
lyre snake	*Trimorphodon biscutatus*	1
night snake	*Hypsiglena torquata*	4
Viperidae		
speckled rattlesnake	*Crotalus mitchelii*	1, 4
sidewinder	*Crotalus cerastes*	1, 4
Mojave rattlesnake	*Crotalus scutulatus*	1, 4

Notes:
* - Sensitive Species occurring on Fort Irwin
s. - subspecies
v. - variety
cf - identification in doubt, based on sterile specimen or no voucher in existence.
2+ - no vouchers made but based on positive identification.

TABLE 2-3

MAMMALS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
CLASS MAMMALIA		
Soricidae		
desert shrew	*Notiosorex crawfordi*	4
Vispertilionidae		
California myotis	*Myotis californicus*	10
western pipistrelle	*Pipistrellus hesperus*	10
big brown bat	*Eptesicus fuscus*	10
Townsend's big-eared bat	*Plecotus townsendii**	10
pallid bat	*Antrozous pallidus**	10
Molassidae		
Mexican free-tailed bat	*Tadarida brasiliensis*	10
western mastiff bat	*Eumops perotis californicus**	10
Bassariscidae		
ring-tailed cat	*Bassariscus astutus*	4+, 10
Mustelidae		
badger	*Taxidea taxus**	4
striped skunk	*Mephitis mephitis*	10
Canidae		
coyote	*Canis latrans*	4
kit fox	*Vulpes macrotis*	4
gray fox	*Urocyon cinereoargenteus*	5
Felidae		
mountain lion	*Felis concolor*	13
bobcat	*Lynx rufus*	4, 10
Sciuridae		
Mohave ground squirrel	*Spermophilus mohavensis**	3, 4
round-tailed ground squirrel	*Spermophilus tereticaudus*	3, 4
antelope ground squirrel	*Ammospermophilus leucurus*	3, 4
Geomydidae		
valley pocket gopher	*Thomomys bottae*	2
Heteromyidae		
little pocket mouse	*Perognathus longimembris*	3, 4
desert pocket mouse	*Perognathus penicillatus*	3, 4
long-tailed pocket mouse	*Chaetodipus formosus*	3, 4
Panamint kangaroo rat	*Dipodomys panamintinus*	3, 4
Great Basin kangaroo rat	*Dipodomys microps*	3, 4
desert kangaroo rat	*Dipodomys deserti*	3, 4
Merriam's kangaroo rat	*Dipodomys merriami*	3, 4
Cricetidae		
cactus mouse	*Peromyscus eremicus*	3, 4
canyon mouse	*Peromyscus crinitus*	3, 4
deer mouse	*Peromyscus maniculatus*	3, 4
brush mouse	*Peromyscus boylei*	3, 4
pinyon mouse	*Peromyscus truei*	3
southern grasshopper mouse	*Onychomys torridus*	2, 3, 4
desert woodrat	*Neotoma lepida*	3, 4
Leporidae		
black-tailed jack rabbit	*Lepus califonicus*	4

TABLE 2-3

MAMMALS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
desert cottontail	*Sylvilagus audubonii*	3, 4
Bovidae		
desert big horn sheep	*Ovis canadensis nelsoni**	4
Equidae		
wild burros	*Equus asinus*	4

Notes:
* - Sensitive Species occurring on Fort Irwin
s - subspecies
v - variety
cf - identification in doubt, based on sterile specimen or no voucher in existence
2+ - no vouchers made but based on positive identification

TABLE 2-4

BIRDS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
BIRDS	**CLASS AVES**	
Podicipedidae		
western grebe	*Aerchmophorus occidentalis**	6
horned grebe	*Podiceps auritus*	6, 7
eared grebe	*Podiceps nigricollis*	6, 7
pied-billed grebe	*Podilymbus podiceps*	6, 7
Ardeidae		
great egret	*Casmerodius albus**	6, 7, 4
snowy egret	*Egretta thula**	7, 4
cattle egret	*Bubulcus ibis*	6, 7
great blue heron	*Ardea herodias**	6
black-crowned night heron	*Nycticorax nycticorax**	4
American bittern	*Botaurus lentiginosus*	4
Threskiornithidae		
white-faced ibis	*Plegadis chihi**	7, 4
Anatidae		
Canada geese	*Branta canadensis*	4
greater white-fronted geese	*Anser fabalis*	4
mallard	*Anas platyrhynchos*	6, 7, 4
Gadwall	*Anas strepera*	6, 7
American widgeon	*Anas americana*	6, 7
northern shoveler	*Anas clypeata*	6, 7
blue-winged teal	*Anas discors*	6, 7
cinnamon teal	*Anas cyanoptera*	6, 7, 4
green-winged teal	*Anas crecca*	6, 7, 4
wood duck	*Aix sponsa*	6, 7
redhead	*Aythya americana*	6, 7
canvasback	*Aythya valisineria*	6, 7
ring-necked duck	*Aythya collaris*	6, 7
lesser scaup	*Aythya affinis*	6, 7
bufflehead	*Bucephala albeola*	6, 7
ruddy duck	*Oxyura jamaicensis*	6, 7, 4
Cathartidae		
turkey vulture	*Cathartes aura*	6,7
Accipitridae		
sharp-shinned hawk	*Accipiter striatus**	6, 7
Copper's hawk	*Accipiter cooperii**	6, 7, 4
northern goshawk	*Accipiter gentilis*	12
northern harrier	*Circus cyaneus**	6, 7, 4
red-tailed hawk	*Buteo jamaicensis*	6, 7, 4
Swainson's hawk	*Buteo swainsoni**	6
golden eagle	*Aquila chrysaetos**	6, 4
Falconidae		
prairie falcon	*Falco mexicanus**	6, 7, 4
American kestrel	*Falco sparverius*	6, 7, 4
Phasianidae		
Gambel's quail	*Callipepla gambelii*	6
chukar (I)	*Alectoris chukar*	6
Rallidae		

TABLE 2-4

BIRDS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
Virginia rail	*Rallus limicola*	6, 7
sora	*Porzana carolina*	6, 7
black rail	*Laterallus jamaicensis**	6, 7
American coot	*Fulica americana*	6, 7, 4
Charadriidae		
semipalmated plover	*Charadrius semipalmatus*	7
killdeer	*Charadrius vociferus*	6, 7
Recurvirostridae		
American avocet	*Recurvirostra americana*	6, 7
black-necked stilt	*Himantopus mexicanus*	6, 7
Scolopacidae		
long-billed curlew	*Numenius americanus**	6
greater yellow legs	*Tringa melanoleuca*	7
spotted sandpiper	*Actitus macularia*	6, 7
western sandpiper	*Calidris pusilla*	7
dunlin	*Calidris alpina*	7
long-billed dowitcher	*Limnodromus griseus*	7
Wilson's phalarope	*Phlaropus tricolor*	7
red-necked phalarope	*Phalaropus lobatus*	6, 7
common snipe	*Gallinago gallinago*	6, 7
Laridae		
California gull	*Larus californicus**	6, 7
ring-billed gull	*Larus delawarensis*	6, 7
Bonaparte's gull	*Larus philadelphia*	6, 7
yellow-footed gull	*Larus livens*	7
black tern	*Chlidonias niger**	7
Columbidae		
mourning dove	Zenaida macroura	6, 7
white-winged dove	Zenaida asiatica	12
ringed turtle-dove (I)	Streptopelia risoria	6
Cuculidae		
greater roadrunner	Geococcyx californianus	6, 7
Tytonidae		
barn owl	*Tyto alba*	6, 4
Strigidae		
western screech owl	*Otus kennicottii*	4
great horned owl	*Bubo virginianus*	6, 4
long-eared owl	*Asio otus**	6, 4
short-eared owl	*Asio flammeus**	12
burrowing owl	*Athene cunicularia**	6,4
Caprimulgidae		
common poorwill	*Phalaenoptilus nuttallii*	12
lesser nighthawk	*Chordeiles acutipennis*	6, 7
Apodidae		
Vaux's swift	*Chaetura vauxi**	6
white-throated swift	*Aeronautes saxatalis*	6, 7
Trochilidae		
black-chinned hummingbird	*Archilochus alexandri*	6
Anna's hummingbird	*Calypte anna*	12

TABLE 2-4

BIRDS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
Costa's hummingbird	*Calypte costae*	6
rufous hummingbird	*Selasphorus rufus*	6
Alcedinidae		
belted kingfisher	*Ceryle alcyon*	6, 7
Picidae		
northern flicker	*Colaptes auratus*	6, 7
ladder-backed woodpecker	*Picoides scalaris*	6
Tyrannidae		
Western kingbird	*Tyrannus verticalis*	6, 7, 4
Cassin's kingbird	*Tyrannus vociferans*	6
ash-throated flycatcher	*Myiarchus cinerascens*	6, 7
black phoebe	*Sayornis nigricans*	6, 7, 4
Say's phoebe	*Sayornis saya*	6, 7, 4
olive-sided flycatcher	*Contopus borealis*	12
western wood-pewee	*Contopus sordidulus*	6, 7
willow flycatcher	*Empidonax trailli*	12
Empidonax spp.	*Empidonax spp.*	6, 7
Alaudidae		
horned larks	*Eremophila alpestris**	6, 7, 4
Hirundinidae		
barn swallow	*Hirundo rustica*	6, 7
cliff swallow	*Hirundo pyrrhonota*	6, 7
violet-green swallow	*Tachycineta thalassina*	6, 7
tree swallow	*Tachycineta bicolor*	6
northern rough-winged swallow	*Stelgidopteryx serripennis*	6, 7
Corvidae		
common raven	*Corvus corax*	6, 7, 4
Remizidae		
verdin	*Auriparus flaviceps*	6, 4
Troglodytidae		
house wren	*Troglodytes aedon*	12
Bewick's wren	*Thryomanes bewickii*	6, 7
cactus wren	*Campylorhynchus brunneicapillus*	6, 4
rock wren	*Salpinctes obsoletus*	6, 4
canyon wren	*Catherpes mexicanus*	6
marsh wren	*Cistothorus palustris*	6, 7
Muscicapidae		
American robin	*Turdus migratorius*	6, 7
hermit thrush	*Catharus guttatus*	6, 7
mountain bluebird	*Sialia currucoides*	6, 7
blue-gray gnatcatcher	*Polioptila caerulea*	6
black-tailed gnatcatcher	*Polioptila nigriceps**	6
ruby-crowned kinglet	*Regulus calendula*	6, 7
Mimidae		
northern mockingbird	*Mimus polyglottos*	6, 7, 4
sage thrasher	*Oreoscoptes montanus*	13
Bendire's thrasher	*Toxostoma bendirei**	6
Le Conte's thrasher	*Toxostoma lecontei**	6, 4
crissal thrasher	*Toxostoma dorsale**	6

TABLE 2-4

BIRDS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
Motacillidae		
American pipit	*Anthus rubescens*	6, 7
Bombycillidae		
cedar waxwing	*Bombycilla cedrorum*	6
Ptilogonatidae		
phainopepla	*Phainopepla nitens*	6
Laniidae		
loggerhead shrike	*Lanius ludovicianus**	6, 7, 4
Sturnidae		
European starling (I)	*Sturnus vulgaris*	6, 7
Vireonidae		
gray vireo	*Vireo vicinior**	6
solitary vireo	*Vireo solitarius*	6, 7
Bell's vireo	*Vireo bellii*	6
least Bell's vireo	*Vireo bellii pusillus**	4
warbling vireo	*Vireo gilvus*	6, 7
Emberizidae		
orange-crowned warbler	*Vermivora celata*	6, 7
Tennessee warbler	*Vermivora peregrina*	8?
Nashville warbler	*Vermivora ruficapilla*	6
Virginia's warbler	*Vermivora virginiae**	6
yellow warbler	*Dendroica petechia**	6, 7
yellow-rumped warbler	*Dendroica coronata*	6, 7
black-throated gray warbler	*Dendroica nigrescens*	6
Townsend's warbler	*Dendroica townsendi*	6, 7
hermit warbler	*Dendroica occidentalis*	6
MacGillivray's warbler	*Oporornis tolmiei*	6, 7
common yellowthroat	*Geothlypis trichas*	6, 7
yellow-breasted chat	*Icteria virens**	6
Wilson's warbler	*Wilsonia pusilla*	6, 7
western tanager	*Piranga lodoviciana*	6, 7
black-headed grosbeak	*Pheucticus melanocephalus*	6, 7
indigo bunting	*Passerina cyanea*	7
lazuli bunting	*Passerina amoena*	6
green-tailed towhee	*Pipilo chlorurus*	6
rufous-sided towhee	*Pipilo erythrophthalmus*	12
American tree sparrow	*Spizella arborea*	6, 7
chipping sparrow	*Spizella passerina*	6, 7
Brewer's sparrow	*Spizella breweri*	6, 4
lark sparrow	*Chindestes grammacus*	6
black-throated sparrow	*Amphispiza bilineata*	6, 4
sage sparrow	*Amphispiza belli*	6, 4
Savannah sparrow	*Passerculus sandwichensis*	6, 7
song sparrow	*Melospiza melodia*	6, 7, 4
Lincoln's sparrow	*Melospiza lincolnii*	6
white-crowned sparrow	*Zonotrichia leucophrys*	6, 7, 4
dark-eyed junco	*Junco hyemalis*	6, 7
red-winged blackbird	*Agelaius phoeniceus*	6, 7

TABLE 2-4

BIRDS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Common Name	Scientific Name	Source
western meadow lark	*Sturnella neglecta*	6, 7
yellow-headed blackbird	*Xanthocephalus xanthocephalus*	6, 7
rusty blackbird	*Euphagus carolinus*	6
Brewer's blackbird	*Euphagus cyanocephalus*	6, 7
brown-headed cowbird	*Molothrus ater*	6, 7
great-tailed grackle	*Quiscalus mexicanus*	6, 7
boat-tailed grackle	*Quiscalus major*	9
northern oriole	*Icterus galbula*	6
Scott's oriole	*Icterus parisorum*	6, 4
Fingillidae		
house finch	*Carpodacus mexicana*	6, 7
lesser goldfinch	*Carduelis psaltria*	6
Passeridae		
house sparrow (I)	*Passer domesticus*	6, 7

Notes:
* - Indicates sensitive species status; see Table 2.2-2
\+ - Unconfirmed sighting, personal communication from Fort Irwin Enviro. Div personnel.
(I) - Introduced species

TABLE 2-4

BIRDS KNOWN TO OCCUR ON FORT IRWIN
FORT IRWIN, CALIFORNIA

Sources:

1 Robert D. Niehaus, Inc., 1995. Herpetological Survey and Physiological Studies on the Western Portion of Fort Irwin NTC.

2 Nagy, K.A. and I L. Brown, 1994. Ecological and Impact Sensitivity of Reptiles at Fort Irwin.

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4 Krzysik, A.J., 1991 Ecological Assessment of Military Training Effects on Threatened, Endangered, and Sensitive Animals and Plants at Fort Irwin, CA.

5 Forrest Shepard and Dr. Bob Rechtman, personal communication

6 Brydolf, Barbara, with Robert D. Hiehaus, Inc. 1996. Final Report, 1994 Avian Survey at the National Training Center, Fort Irwin, CA.

7 Brydolf, Barbara, 1995. Birds Found at Fort Irwin Sewage Treatment Ponds, 1994 and Spring 1995.

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10 Brown, Patricia E., 1994. Bat Survey of the NTC Fort Irwin, San Bernardino Co , CA.

11 Morafka, David J., 1993. Amphibian and Reptile Study of 20 Sites at the NTC, Fort Irwin. (Draft Report).

12 McCalvin, Catherine and David Pereksta, 1994. Biological Inventory of the Hellwind Conyon Drainage System, Military Fort Irwin Reservation, San Bernardino Co. CA

13 Steve Ahman, personal communication

TABLE 5-1

BACKGROUND SUMMARY STATISTICS FOR SOILS[a]
FORT IRWIN, CALIFORNIA

Constituent	n	%nd	Range of SQLs for Non-detects[b]			min (mg/kg)	max (mg/kg)	mean (mg/kg)	median (mg/kg)	s	CV	BUTL (mg/kg)
Aluminum	119	0	NA	-	NA	2,920	29,900	9,790	8,420	4,980	0.509	23,600
Antimony	55	0	NA	-	NA	1.54	6.46	2.74	2.11	1.26	0.46	6.34
Arsenic	119	0	NA	-	NA	0.642	26.3	2.94	2.18	2.9	0.986	9.14
Barium	119	0	NA	-	NA	13.9	252	65.3	58.5	34	0.521	175
Beryllium	119	0	NA	-	NA	0.171	1.88	0.482	0.43	0.249	0.517	1.17
Cadmium	119	16.8	0.408	-	0.443	0.0417	0.668	0.184	0.193	0.109	0.594	0.416
Chromium	119	0	NA	-	NA	1.61	63.3	9.94	6.65	8.62	0.867	27.7
Cobalt	119	0	NA	-	NA	0.814	13.9	4.88	4.14	2.79	0.572	12.9
Copper	119	0	NA	-	NA	1.77	34.8	10.4	7.54	6.83	0.66	28.7
Hexavalent Chromium	59	96.6	0.218	-	1	0.109	0.62	0.398	0.392	0.0613	0.154	0.512
Lead	119	0	NA	-	NA	0.988	9.49	3.83	3.72	1.41	0.369	7.33
Manganese	118	0	NA	-	NA	80.3	402	203	200	66.2	0.326	361
Mercury	119	95.8	0.169	-	0.445	0.0587	0.39	0.129	0.0915	0.0511	0.397	0.202
Molybdenum	15	0	NA	-	NA	1.15	3.5	1.87	1.66	1.777	0.416	4.58
Nickel	119	0	NA	-	NA	1.34	35.9	8.24	7.1	5.94	0.721	29.1
Nitrate/Nitrite-Nitrogen	119	6.7	0.228	-	0.458	0.114	10.8	1.27	0.843	1.56	1.23	632
Selenium	1	0	NA	-	NA	0.553	0.553	0.553	0.553	NA	NA	NA
Silver	1	0	NA	-	NA	0.186	0.186	0.186	0.186	NA	NA	NA
Thallium	83	49.4	3.47	-	3.83	2.05	2.05	1.42	1.74	0.417	0.294	1.91
Vanadium	119	0	NA	-	NA	166	166	25.6	18.9	22	0.856	97
Zinc	119	0	NA	-	NA	79.2	79.2	29.2	26.4	11.3	0.385	51

Notes:

[a] One-half the reporting limit was used to calculate the summary statistics.

[b] Range of SQLs represent non-detects that were included in the data set; data with SQLs greater than 2 times the maximum site concentration are not included in the data set.

%nd - percentage of samples reported as non-detects
BUTL - background upper tolerance limit
CV - coefficient of variation
max - maximum detected value
mg/kg - milligram per kilogram
min - minimum detected value
n - number of samples
NA - not applicable because there are 0 non-detects for these analytes
s - standard deviation
SQL - sample quantitation limit

TABLE 5-2

SELECTION OF CHEMICALS OF POTENTIAL CONCERN FOR SURFACE SOILS [a]
SITE FTIR-32A
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration (mg/kg) Maximum [b]		Minimum [c]		Mean [d]	Number of Samples [e]	Number of Detections [f]	Detection Frequency	BUTL [g] (mg/kg)	COPC [h]
Inorganics										
Aluminum	14,745		5,460		8,195	18	18	100%	23,600	No
Antimony	0.98	J	0.135	nd	0.301	18	6	33%	6.34	No
Arsenic	9.8		4.1	J	5.7	18	18	100%	9.14	Yes
Barium	182		84.3		115.1	18	18	100%	175	Yes
Beryllium	1.1	J	0.27	J	0.51	18	18	100%	1.17	No
Cadmium	1.5	J	0.079	J	0.317	18	18	100%	0.416	Yes
Calcium	24,200		9.2		6,937	18	18	100%	na	No [j]
Chromium	12.4		4.4		7.1	18	18	100%	27.7	No
Cobalt	8.8	J	4.2	J	6.0	18	18	100%	12.9	No
Copper	419		10.8		44.6	18	18	100%	28.7	Yes
Iron	16,700		6,260		10,448	18	18	100%	na	No [j]
Lead	58.7		9.9		15.8	18	18	100%	7.33	Yes
Magnesium	7,780		3,690		4,678	18	18	100%	na	No [j]
Manganese	649		363		482	18	18	100%	361	Yes
Molybdenum	0.24	J	0.0545	nd	0.105	18	7	39%	4.58	No
Nickel	12.7	J	5.4	J	7.6	18	18	100%	29.1	No
Potassium	4,100		1,370		2,164	18	18	100%	na	No [j]
Selenium	2.3	J	0.69	J	1.52	18	18	100%	na	Yes
Sodium	132	J	24.1	nd	39.4	18	5	28%	na	No [j]
Thallium	0.8	J	0.187	nd	0.283	18	4	22%	1.91	No
Vanadium	23.2		10.8		16.5	18	18	100%	97	No
Zinc	1,500		34.2		210	18	18	100%	51	Yes
VOC										
Tetrachloroethylene	0.010		0.0025	nd	0.003	18	2	11%	na	Yes
Toluene	0.0009	J	0.0025	nd	0.0025	18	1	6%	na	Yes
Trichloroethylene	0.003	J	0.0025	nd	0.0025	18	4	22%	na	Yes

TABLE 5-2

SELECTION OF CHEMICALS OF POTENTIAL CONCERN FOR SURFACE SOILS [a]
SITE FTIR-32A
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration (mg/kg)			Number of		Detection	BUTL [g]	
	Maximum [b]	Minimum [c]	Mean [d]	Samples [e]	Detections [f]	Frequency	(mg/kg)	COPC [h]
TPH								
TRPH	57	16 [i]	nd 18	18	3	17%	na	Yes

Notes:

[a] Surface soil depth equal to 0 - 1 foot bgs.

[b] Maximum detected concentration of original or duplicate soil samples (0 - 1 foot bgs).

[c] Minimum concentration of original or duplicate samples (0 - 1 foot bgs).

[d] The arithmetic mean soil concentration for soil samples collected in the interval 0 - 1 foot bgs.

[e] Total number of soil samples collected in the interval 0 - 1 foot bgs (excludes duplicate samples).

[f] Total number of detections in soil samples collected in the interval 0 - 1 foot bgs (excludes duplicate samples).

[g] Background upper tolerance limit for Fort Irwin Soils. Source: Parsons Engineering Science, Inc., 1996.

[h] Constituent is considered a chemical of potential concern (COPC) if maximum concentration exceeds BUTL.

[i] Minimum concentration is a non-detect value and is assumed to be one-half the detection limit.

[j] Analyte eliminated as a COPC based on its role as an essential dietary nutrient (refer to Section 5.2.4).

J - estimated value
mg/kg - milligrams per kilogram
na - not applicable
nd - not detected
TPH - Total petroleum hydrocarbons
TRPH - Total recoverable petroleum hydrocarbons
VOC - Volatile organic compounds

TABLE 5-3

SELECTION OF CHEMICALS OF POTENTIAL CONCERN FOR SUBSURFACE SOILS [a]
SITE FTIR-32A
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration (mg/kg) Maximum [b]		Minimum [c]		Mean [d]	Number of Samples [e]	Number of Detections [f]	Detection Frequency	BUTL [g] (mg/kg)	COPC [h]
Inorganics										
Aluminum	8,630		4,100		6,886	18	18	100%	23,600	No
Antimony	3.1	J	0.137 [i]	nd	0.63	18	5	28%	6.34	No
Arsenic	35.6		4.1	J	7.9	18	18	100%	9.14	Yes
Barium	384		62.5		133.5	18	18	100%	175	Yes
Beryllium	0.73	J	0.29	J	0.47	18	18	100%	1.17	No
Cadmium	4.9		0.045 [i]	nd	0.81	18	17	94%	0.416	Yes
Calcium	16,400		5,730		9,604	18	18	100%	na	No [j]
Chromium	21.5		3.8	J	8.1	18	18	100%	27.7	No
Cobalt	12.2		4	J	6	18	18	100%	12.9	No
Copper	2,360		12.4		288	18	18	100%	28.7	Yes
Iron	16,200		5,000		9,607	18	18	100%	na	No [j]
Lead	103		7.7		30	18	18	100%	7.33	Yes
Magnesium	5,130		2,710		3,679	18	18	100%	na	No [j]
Manganese	723		331		436	18	18	100%	361	Yes
Mercury	0.31		0.05 [i]	nd	0.074	18	3	17%	0.202	Yes
Molybdenum	0.45	J	0.056 [i]	nd	0.126	18	7	39%	4.58	No
Nickel	11.2	J	4.4	J	6.8	18	18	100%	29.1	No
Potassium	2,510	J	995		1,531	18	18	100%	na	No [j]
Selenium	2.2	J	0.93	J	1.4	18	16	89%	na	Yes
Sodium	321		24.5 [i]	nd	72.6	18	8	44%	na	No [j]
Thallium	1.4	J	0.19 [i]	nd	0.4	18	7	39%	1.91	No
Vanadium	22		9.3		13.6	18	18	100%	97	No
Zinc	3,230		25.5	J	539	18	18	100%	51	Yes
VOC										
1,2-Dichlorobenzene	0.028	J	0.085 [i]	nd	0.086	18	1	6%	na	Yes
1,3-Dichlorobenzene	0.062	J	0.085 [i]	nd	0.088	18	1	6%	na	Yes
Tetrachloroethylene	0.002	J	0.0025 [i]	nd	0.0026	18	1	6%	na	Yes
Trichloroethylene	0.003	J	0.0025 [i]	nd	0.0027	18	2	11%	na	Yes

TABLE 5-3

SELECTION OF CHEMICALS OF POTENTIAL CONCERN FOR SUBSURFACE SOILS [a]
SITE FTIR-32A
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration (mg/kg) Maximum [b]		Minimum [c]		Mean [d]	Number of Samples [e]	Detections [f]	Detection Frequency	BUTL [g] (mg/kg)	COPC [h]
SVOC										
Benzo(a)anthracene	0.014	J	0.085 [i]	nd	0.086	18	1	6%	na	Yes
Benzo(a)pyrene	0.065	J	0.085 [i]	nd	0.089	18	1	6%	na	Yes
Benzo(b)fluoranthene	0.019	J	0.085 [i]	nd	0.860	18	1	6%	na	Yes
bis(2-Ethylhexyl)phthalate	1.1	J	0.085 [i]	nd	0.145	18	2	11%	na	Yes
Chrysene	0.025	J	0.085 [i]	nd	0.082	18	2	11%	na	Yes
Di-n-butyl phthalate	0.094	J	0.085 [i]	nd	0.090	18	1	6%	na	Yes
Fluoranthene	0.025	J	0.085 [i]	nd	0.079	18	3	17%	na	Yes
Hexachlorobenzene	0.09	J	0.085 [i]	nd	0.09	18	1	6%	na	Yes
Hexachloroethane	0.09	J	0.085 [i]	nd	0.09	18	1	6%	na	Yes
Naphthalene	0.045	J	0.085 [i]	nd	0.085	18	2	11%	na	Yes
Phenanthrene	0.06	J	0.085 [i]	nd	0.081	18	3	17%	na	Yes
Pyrene	0.061	J	0.085 [i]	nd	0.088	18	1	6%	na	Yes
1,2,4-Trichlorobenzene	0.042	J	0.085 [i]	nd	0.083	18	2	11%	na	Yes
TPH										
TRPH	82		15.5 [i]	nd	23	18	4	22%	na	Yes
Dioxins/Furans										
2,3,7,8-TCDD	0.000011		0.000011		0.000011	1	1	100%	na	Yes
Total TCDD	0.001100		0.0011		0.0011	1	1	100%	na	Yes
1,2,3,7,8-PeCDD	0.000058		0.000058		0.000058	1	1	100%	na	Yes
Total PeCDD	0.001400		0.0014		0.0014	1	1	100%	na	Yes
1,2,3,4,7,8-HxCDD	0.000080		0.00008		0.00008	1	1	100%	na	Yes
1,2,3,6,7,8-HxCDD	0.000130		0.00013		0.00013	1	1	100%	na	Yes
1,2,3,7,8,9-HxCDD	0.000075		0.000075		0.000075	1	1	100%	na	Yes
Total HxCDD	0.002100		0.0021		0.0021	1	1	100%	na	Yes
1,2,3,4,6,7,8-HpCDD	0.000790		0.00079		0.00079	1	1	100%	na	Yes
Total HpCDD	0.001700		0.0017		0.0017	1	1	100%	na	Yes
OCDD	0.001300		0.0013		0.0013	1	1	100%	na	Yes
2,3,7,8-TCDF	0.000066		0.000066		0.000066	1	1	100%	na	Yes
Total TCDF	0.003000		0.003		0.003	1	1	100%	na	Yes
1,2,3,7,8-PeCDF	0.000081		0.000081		0.000081	1	1	100%	na	Yes
2,3,4,7,8-PeCDF	0.000200		0.0002		0.0002	1	1	100%	na	Yes

TABLE 5-3

SELECTION OF CHEMICALS OF POTENTIAL CONCERN FOR SUBSURFACE SOILS [a]
SITE FTIR-32A
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration (mg/kg)			Number of		Detection	BUTL [g]	
	Maximum [b]	Minimum [c]	Mean [d]	Samples [e]	Detections [f]	Frequency	(mg/kg)	COPC [h]
Dioxins/Furans (cont'd)								
Total PeCDF	0.002400	0.0024	0.0024	1	1	100%	na	Yes
1,2,3,4,7,8-HxCDF	0.000160	0.00016	0.00016	1	1	100%	na	Yes
1,2,3,6,7,8-HxCDF	0.000170	0.00017	0.00017	1	1	100%	na	Yes
2,3,4,6,7,8-HxCDF	0.000300	0.0003	0.0003	1	1	100%	na	Yes
1,2,3,7,8,9-HxCDF	0.000059	0.000059	0.000059	1	1	100%	na	Yes
Total HxCDF	0.002000	0.002	0.002	1	1	100%	na	Yes
1,2,3,4,6,7,8-HpCDF	0.000690	0.00069	0.00069	1	1	100%	na	Yes
1,2,3,4,7,8,9-HpCDF	0.000054	0.000054	0.000054	1	1	100%	na	Yes
Total HpCDF	0.000970	0.00097	0.00097	1	1	100%	na	Yes
OCDF	0.000170	0.00017	0.00017	1	1	100%	na	Yes
TEQ (2,3,7,8-TCDD)	0.00020							

Notes:
[a] Soil depth equal to >1 - 10 feet bgs
[b] Maximum detected concentration of original or duplicate soil samples (1 - 10 feet bgs).
[c] Minimum concentration of original or duplicate samples (1 - 10 feet bgs)
[d] The arithmetic mean soil concentration for soil samples collected in the interval 1 - 10 feet bgs
[e] Total number of soil samples collected in the interval 1 - 10 feet bgs (excludes duplicate samples
[f] Total number of detections in soil samples collected in the interval 1 - 10 feet bgs (excludes duplicate samples).
[g] Background upper tolerance limit for Fort Irwin Soils. Source: Parsons Engineering Science, Inc., 199(
[h] Constituent is considered a chemical of potential concern (COPC) if maximum concentration exceeds BUTL
[i] Minimum concentration is a non-detect value and is assumed to be one-half the detection limit
[j] Analyte eliminated as a COPC based on its role as an essential dietary nutrient (refer to Section 5.2.4

J - estimated value
mg/kg - milligrams per kilogram
na - not applicable
nd - not detected
SVOC - Semivolatile organic compounds
TEQ (2,3,7,8-TCDD) - 2,3,7,8-TCDD toxicity equivalent concentration
TPH - Total petroleum hydrocarbons
TRPH - Total recoverable petroleum hydrocarbons
VOC - Volatile organic compounds

TABLE 5-4

SUMMARY OF CHEMICALS OF POTENTIAL CONCERN (COPC) [a]
SITE FTIR-32A
FORT IRWIN, CALIFORNIA

Surface Soils [b]	Subsurface Soils [c]	
Inorganics	**Inorganics**	**Dioxins/Furans**
Arsenic	Arsenic	2,3,7,8-TCDD
Barium	Barium	Total TCDD
Cadmium	Cadmium	1,2,3,7,8-PeCDD
Copper	Copper	Total PeCDD
Lead	Lead	1,2,3,4,7,8-HxCDD
Manganese	Manganese	1,2,3,6,7,8-HxCDD
Selenium	Mercury	1,2,3,7,8,9-HxCDD
Zinc	Selenium	Total HxCDD
	Zinc	1,2,3,4,6,7,8-HpCDD
		Total HpCDD
VOC	**VOC**	OCDD
Tetrachloroethylene	1,2-Dichlorobenzene	2,3,7,8-TCDF
Toluene	1,3-Dichlorobenzene	Total TCDF
Trichloroethylene	Tetrachloroethylene	1,2,3,7,8-PeCDF
	Trichloroethylene	2,3,4,7,8-PeCDF
		Total PeCDF
TPH	**SVOC**	1,2,3,4,7,8-HxCDF
TRPH	Benzo(a)anthracene	1,2,3,6,7,8-HxCDF
	Benzo(a)pyrene	2,3,4,6,7,8-HxCDF
	Benzo(b)fluoranthene	1,2,3,7,8,9-HxCDF
	bis(2-Ethylhexyl)phthalate	Total HxCDF
	Chrysene	1,2,3,4,6,7,8-HpCDF
	Di-n-butyl phthalate	1,2,3,4,7,8,9-HpCDF
	Fluoranthene	Total HpCDF
	Hexachlorobenzene	OCDF
	Hexachloroethane	
	Naphthalene	
	Phenanthrene	
	Pyrene	**TPH**
	1,2,4-Trichlorobenzene	TRPH

Notes:

[a] Refer to Tables 5-2 and 5-3 for COPC selection.

[b] Surface soil depth equal to 0 - 1 foot bgs.

[c] Subsurface soil depth > 1 - 10 feet bgs.

COPC - Chemical of potential concern
SVOC - Semivolatile organic compounds
TPH - Total petroleum hydrocarbons
TRPH - Total recoverable petroleum hydrocarbons
VOC - Volatile organic compounds

TABLE 5-5

EXPOSURE POINT CONCENTRATION DETERMINATION FOR SURFACE SOILS
SITE FTIR-32A
FORT IRWIN, CALIFORNIA

Constituent	Maximum Concentration (mg/kg)		Minimum Concentration[a] (mg/kg)		95% UCL Concentration[b] (mg/kg)	Exposure Point Concentration[c] (mg/kg)
Metals						
Arsenic	9.8		4.1	J	6.3	6.3
Barium	182		84.3		125	125
Cadmium	1.5	J	0.079	J	0.5	0.5
Copper	419		10.8		57	57
Lead	58.7		9.9		18.5	18.5
Manganese	649		363		507	507
Selenium	2.3	J	0.69	J	1.7	1.7
Zinc	1,500		34.2		328	328
VOC						
Tetrachloroethylene	0.01		0.0025	nd	0.004	0.004
Toluene	0.0009	J	0.0025	nd	nc	0.0009
Trichloroethylene	0.003	J	0.0025	nd	0.003	0.003
TPH						
TRPH	57		16	nd	20	20

Notes:

[a] Minimum detected concentration or one half of the reporting limit(marked nd)

[b] The 95% upper confidence limit of all results, using one half of the detection limit for non-detect results.

[c] The lower value of the maximum detected concentration or the UCL concentration.

J-Estimated

mg/kg - milligrams per kilogram

nc-Not calculated

nd - Not detected, value represents one half of the reporting limit.

TPH - Total petroleum hydrocarbons

TRPH - Total recoverable petroleum hydrocarbons

UCL - Upper Confidence Limit.

VOC - Volatile organic compounds

TABLE 5-6

EXPOSURE POINT CONCENTRATION DETERMINATION FOR SUBSURFACE SOILS
SITE FTIR-32A
FORT IRWIN, CALIFORNIA

Constituent	Maximum Concentration (mg/kg)		Minimum Concentration[a] (mg/kg)		95% UCL Concentration[b] (mg/kg)	Exposure Point Concentration[c] (mg/kg)
Metals						
Arsenic	35.6		4.1	J	9.9	9.9
Barium	384		62.5		167	167
Cadmium	4.9		0.045	nd	2.5	2.5
Copper	2,360		12.4		1,386	1,386
Lead	103		7.7		50	50
Manganese	723		331		471	471
Mercury	0.31		0.05	nd	0.09	0.09
Selenium	2.2	J	0.93	J	1.5	1.5
Zinc	3,230		25.5	J	3,263	3,230
VOC						
1,2-Dichlorobenzene	0.028	J	0.085	nd	nc	0.028
1,3-Dichlorobenzene	0.062	J	0.085	nd	nc	0.062
Tetrachloroethylene	0.002	J	0.0025	nd	nc	0.002
Trichloroethylene	0.003	J	0.0025	nd	0.003	0.003
SVOC						
Benzo(a)anthracene	0.014	J	0.085	nd	nc	0.014
Benzo(a)pyrene	0.065	J	0.085	nd	nc	0.065
Benzo(b)fluoranthene	0.019	J	0.085	nd	nc	0.019
bis(2-Ethylhexyl)phthalate	1.1	J	0.085	nd	0.2	0.2
Chrysene	0.025	J	0.085	nd	nc	0.025
Di-n-butyl phthalate	0.094	J	0.085	nd	0.09	0.09
Fluoranthene	0.025	J	0.085	nd	nc	0.025
Hexachlorobenzene	0.09	J	0.085	nd	0.09	0.09
Hexachloroethane	0.09	J	0.085	nd	0.09	0.09

TABLE 5-6

EXPOSURE POINT CONCENTRATION DETERMINATION FOR SUBSURFACE SOILS
SITE FTIR-32A
FORT IRWIN, CALIFORNIA

Constituent	Maximum Concentration (mg/kg)		Minimum Concentration[a] (mg/kg)		95% UCL Concentration[b] (mg/kg)	Exposure Point Concentration[c] (mg/kg)
Naphthalene	0.045	J	0.085	nd	nc	0.045
Phenanthrene	0.060	J	0.085	nd	nc	0.060
Pyrene	0.061	J	0.085	nd	nc	0.061
1,2,4-Trichlorobenzene	0.042	J	0.085	nd	nc	0.042
Dioxins/Furans						
TEQ (2,3,7,8)-TCDD	0.00020		0.00020		nc	0.00020
TPH						
TRPH	82		15.5	nd	29	29

Notes:

[a] Minimum detected concentration or one half of the reporting limit(marked nd)

[b] The 95% upper confidence limit of all results, using one half of the detection limit for non-detect results.

[c] The lower value of the maximum detected concentration or the UCL concentration.

J-Estimated
mg/kg - milligrams per kilogram
nc-Not calculated
nd - Not detected, value represents one half of the reporting limit.
SVOC - Semivolatile organic compounds
TPH - Total petroleum hydrocarbons
TRPH - Total recoverable petroleum hydrocarbons
UCL - Upper Confidence Limit.
VOC - Volatile organic compounds

TABLE 5-7

EXPOSURE PARAMETERS AND ASSUMPTIONS FOR MILITARY PERSONNEL
FORT IRWIN, CALIFORNIA

Exposure Parameter	Units	Exposure Assumptions: Normal Activity [a]	Exposure Assumptions: Moderate-High Activity [b]	Source
Air Concentration - C_a	ug/m^3	Chemical-specific	Chemical-specific	Not applicable
Soil Concentration - C_s	mg/kg	Chemical-specific	Chemical-specific	Not applicable
Body Weight - BW	kg	70	70	USEPA, 1997
Soil Ingestion Rate - IR	mg/day	75 [c]	720 [d]	Parsons, 1995
Inhalation Rate - InhR	m^3/hr	0 83	2 5	USEPA, 1997
Exposure Time - ET	hr/day	12 [e]	12 [e]	Parsons, 1995
Exposure Frequency - EF	day/yr	154 [f]	154 [f]	Parsons, 1995
Exposure Duration - ED	yr	4 [g]	4 [g]	Parsons, 1995
Dermal Surface Area - SA	cm^2/event	4,300 [h]	4,300 [h]	Parsons, 1995
Skin Adherence Factor - AF	mg/cm^2	1	1	Parsons, 1995
Particulate Emission Factor - PEF	m^3/kg	1 6E+07 [i]	3 3E+06 [j]	Parsons, 1995
Averaging Time - AT	days			
Carcinogens		25,550	25,550	USEPA, 1989
Noncarcinogens		1460 [k]	1460 [k]	Parsons, 1995

Sources:
Exposure Factors Handbook (USEPA, 1997).
Project Workplan for the Site Inspection and Remedial Investigation of 31 Sites at the National Training Center Fort Irwin, California (Parsons 1995)
Risk Assessment Guidance for Superfund (RAGS) Volume 1: Human Health Evaluation manual (Part A) (USEPA 1989).

Notes:
[a] Assumptions are based on a military worker engaged in light work or at rest.
[b] Assumptions are based on a military worker engaged in moderate to heavy work while conducting military maneuvers
[c] Assumes an incidental soil ingestion rate of 6 25 mg/hr (50 mg/day / 8 hr) over a period of 12 hours.
[d] Assumes an incidental soil ingestion rate of 60 mg/hr (480 mg/day / 8 hr) over a period of 12 hours.
[e] Assumes a military worker is engaged in light work or at rest for 12 hours and performing moderate to heavy work for 12 hours
[f] Assumes a military worker is engaged in field activities for 14 days per month for 11 months.
[g] Assumes a military worker is stationed at Fort Irwin for a maximum of 4 years.
[h] Assumes exposure of the head, arms, and hands consistent with military attire.
[i] Assumes the highest annual average respirable particulate concentration (61 ug/m^3) reported over the last 4 years for the Mojave Desert Air Quality Management District (in Victorville California)
[j] Assumes a respirable particulate concentration of 305 ug/m^3 for heavy vehicular traffic
[k] Assumption is based on a total exposure duration of 4 years (4 years x 365 days/yr = 1,460 days).

cm^2/event-Square centimeters per event
day/yr-Days per year
hr/day-Hours per day
kg-Kilogram
m^3/hr-Cubic meters per hour
m^3/kg-Cubic meters per kilogram
mg/cm^2-Milligrams per square centimeter
mg/day-Milligrams per day
mg/kg-Milligrams per kilogram
ug/m^3-Micrograms per cubic meter
yr-Year

TABLE 5-8

EXPOSURE PARAMETERS AND ASSUMPTIONS FOR AN INDUSTRIAL WORKER
FORT IRWIN, CALIFORNIA

Exposure Parameter	Units	Exposure Assumption	Source
Soil Concentration - C_s	mg/kg	Chemical-specific	Not applicable
Body Weight - BW	kg	70	USEPA, 1997
Soil Ingestion Rate - IR	mg/day	100	USEPA, 1997
Inhalation Rate - InhR	m^3/hr	2 5	USEPA, 1997
Exposure Time - ET	hr/day	8	USEPA, 1997
Exposure Frequency - EF	day/yr	250	USEPA, 1997
Exposure Duration - ED	yr	25	USEPA, 1997
Dermal Surface Area - SA	cm^2/event	3,300	USEPA, 1999a
Skin Adherence Factor - AF	mg/cm^2	0 2	USEPA, 1999a
Particulate Emission Factor - PEF	m^3/kg	1 6E+07	Parsons ES, 1995
Averaging Time - AT	days		
Carcinogens		25,550	USEPA, 1989
Noncarcinogens		9,125	USEPA, 1989

Sources:
Exposure Factors Handbook (USEPA 1997)
Project Workplan for the Site Inspection and Remedial Investigation of 31 Sites at NTC Fort Irwin (Parsons ES, 1995)
RAGS, Supplemental Guidance-Dermal Risk Assessment (USEPA, 1999a)
Region 9 Preliminary Remediation Goals (PRGs) 1999 (USEPA, 1999b)
Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part A) (USEPA, 1989)

cm^2/event-Square centimeters per event
day/yr-Days per year
hr/day-Hours per day
kg-Kilogram
m^3/hr-Cubic meters per hour
m^3/kg-Cubic meters per kilogram
mg/cm^2-Milligrams per square centimeter
mg/day-Milligrams per day
mg/kg-Milligrams per kilogram
yr-Year

TABLE 5-9

TOXICITY VALUES FOR HUMAN HEALTH RISK ASSESSMENT
FORT IRWIN, CALIFORNIA

Constituent	Cancer Slope Factor - CSF (mg/kg-d)$^{-1}$				Reference Dose - RfD (mg/kg-d)			
	Oral		Inhalation		Oral		Inhalation	
Inorganics								
Arsenic	1.5E+00	I	1.5E+01	I	3.0E-04	I	3.0E-04	R
Barium	NA		NA		7.0E-02	I	1.4E-04	H
Cadmium	NA		6.3E+00	I	5 0E-04	I	5.0E-04	R
Cobalt	NA		NA		6 0E-02	N	6.0E-02	R
Copper	NA		NA		3 7E-02	H	3.7E-02	R
Lead	na		na		na		na	
Manganese	NA		NA		2 4E-02	I	1.4E-05	I
Mercury	NA		NA		3 0E-04	I	3.0E-04	R
Molybdenum	NA		NA		5 0E-03	H	5.0E-03	R
Selenium	NA		NA		5.0E-03	I	5 0E-03	R
Zinc	NA		NA		3.0E-01	I	3 0E-01	R
VOC								
1,2-Dichlorobenzene	NA		NA		9.0E-02	I	5 7E-02	H
1,3-Dichlorobenzene	NA		NA		9.0E-04	N	9.0E-04	R
Tetrachloroethylene	5.2E-02	N	2 0E-03	N	1.0E-02	I	1.1E-01	N
Toluene	NA		NA		2.0E-01	I	1.1E-01	H
Trichloroethylene	1 1E-02	N	6 0E-03	N	6.0E-03	N	6.0E-03	R
SVOC								
Benzo(a)anthracene	7.3E-01	N	3 1E-01	N	na		na	
Benzo(a)pyrene	7.3E+00	I	3.1E+00	N	na		na	
Benzo(b)fluoranthene	7.3E-01	N	3 1E-01	N	na		na	
bis(2-Ethylhexyl)phtha	1.4E-02	I	1.4E-02	R	2 0E-02	I	2.2E-02	R
Chrysene	7.3E-03	N	3.1E-03	N	na		na	
Di-n-butylphthalate	NA		NA		1 0E-01	I	1.0E-01	R
Fluoranthene	NA		NA		4 0E-02	I	4 0E-02	I
Hexachlorobenzene	1.6E+00	I	1.6E+00	I	8 0E-04	I	8 0E-04	R
Hexachloroethane	1.4E-02	I	1.4E-02	I	1 0E-03	I	1 0E-03	R
Naphthalene	NA		NA		2 0E-02	I	8 6E-04	I
Phenanthrene	NA		NA		na		na	
Pyrene	NA		NA		3 0E-02	I	3 0E-02	R
1,2,4-Trichlorobenzene	NA		NA		1.0E-02	I	5.7E-02	H
Dioxins/Furans								
2,3,7,8-TCDD	1 5E-05	H	1 5E-05	H	na		na	
TPH								
TRPH	na		na		na		na	

Notes:

CSF - Cancer slope factor

H - HEAST (USEPA, 1995).

I - IRIS Database (USEPA 2000)

N- National Center for Environmental Assessment

NA - Not applicable.

na - Not available.

R - Route extrapolation.

RfD - Reference dose

SVOC - Semivolatile organic compounds

TPH - Total petroleum hydrocarbons

TRPH - Total residual petroleum hydrocarbons

VOC - Volatile organic compounds

TABLE 5-10

SUMMARY OF BASELINE HUMAN HEALTH RISK ASSESSMENT RESULTS
SITE FTIR-32A
FORT IRWIN, CALIFORNIA

Medium/Exposure Scenario	Total Cancer Risk [a]	Hazard Index (HI) [b]	Primary COPCs [c]	Chemical-Specific Cancer Risk/ Hazard Quotient [d]	Exposure Point Concentration [e] (mg/kg)	Fort Irwin Soil BUTL[f] (mg/kg)	California Ambient Soil[g] (mg/kg)
Surface Soils							
Current/future Military Personnel	3.0E-06	2.4	Arsenic	Risk = 3.0E-06	6.3	9.14	0.6 - 11
			Manganese	HQ = 2.2	507	361	253 - 1,687
Future Industrial Worker	4.4E-06	0.51	Arsenic	Risk = 4.0E-06	6.3	9.14	0.6 - 11
Subsurface Soils							
Current/future Military Personnel	NA	NA	NA	NA	NA	NA	NA
Future Industrial Worker	7.3E-06	0.55	Arsenic	Risk = 6.2E-06	9.9	9.14	0.6 - 11

Notes:

Shading indicates exceedance of the risk or hazard criterion.

[a] Total site-specific cancer risk for the indicated medium.

[b] Total site-specific hazard index for the indicated medium.

[c] The chemicals of potential concern that result in an exceedance of the total risk or hazard criterion

[d] The chemical-specific cancer risk or noncancer hazard quotient that results in an exceedance of the total risk or hazard criterion.

[e] Maximum or 95% UCL concentration detected in the indicated medium.

[f] The background upper tolerance limit (BUTL) for NTC Fort Irwin, California soils.

[g] The range of background concentrations measured in California soils (Source: Bradford et al., 1996)

COPC	Chemical of potential concern.
mg/kg	Milligram per kilogram.
NA	Not applicable.

TABLE 7-1

SUMMARY RESULTS AND CONCLUSIONS
SITES FTIR-32A AND FTIR-39
FORT IRWIN, CALIFORNIA

Risk Endpoint/Exposure Scenario	Site FTIR-32A		Site FTIR-39	
	Surface Soil	Subsurface Soil	Surface Soil	Subsurface Soil
Human Health Risk Assessment Results				
Total Cancer Risk				
Current/Future Military Personnel	3.0E-06	NA[a]	NA[b]	NA[c]
Future Industrial Worker	4.1E-06	6.6E-06	NA[b]	NA[c]
Hypothetical Future Resident[d]	1.7E-05	2.8E-05	1.0E-08	NA[c]
USEPA Acceptable Risk Range[e]:	1.0E-04 - 1.0E-06	1.0E-04 - 1.0E-06	1.0E-04 - 1.0E-06	1.0E-04 - 1.0E-06
Primary COPCs:	Arsenic	Arsenic, B(a)P	NA	NA
Total Hazard Index (HI)				
Current/Future Military Personnel	**2.4**	NA[a]	NA[b]	NA[c]
Future Industrial Worker	0.11	0.12	NA[b]	NA[c]
Hypothetical Future Resident[d]	1.0	0.73	0.82	NA[c]
USEPA Acceptable Risk Range[e]:	1.0	1.0	1.0	1.0
Primary COPCs:	Manganese	NA	NA	NA
Ecological Risk Assessment Results				
Total Hazard Index (HI)				
Mojave Ground Squirrel	NA[f]	NA[f]	NA[f]	NA[f]
Golden Eagle	NA[f]	NA[f]	NA[f]	NA[f]
USEPA Acceptable Risk Range[e]:	1.0	1.0	1.0	1.0
Primary COPECs:	NA	NA	NA	NA
Conclusions				
Site Recommendation:	**NFA**	**NFA**	**NFA**	**NFA**

Notes:
Bolding indicates exceedence of a risk criterion
[a] Potential exposure to subsurface soils was deemed to represent an incomplete pathway for current/future military personnel (refer to Section 5.3.3)
[b] Risks for current/future military personnel and future industrial workers were not quantified in the baseline risk assessment screening because Site FTIR-39 surface soils were within acceptable screening risk criteria. (Please refer to the SI Report; Montgomery Watson, 1998).
[c] Subsurface soils at Site FTIR-39 were not investigated consistent with the surficial nature of the suspected sources
[d] Baseline risk assessment results for the hypothetical future resident for Site FTIR-32A are presented in Appendix E. Please refer to the SI Report (Montgomery Watson 1998) for screening residential risk estimates for Site FTIR-39
[e] Source: Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions (USEPA 1991a).
[f] Sites FTIR-32A and FTIR-39 were determined to contain no useable habitat for ecological receptors (refer to Section 6.0) Therefore, ecological risks for representative indicator species were not quantified for these sites.

B(a)P - Benzo(a)pyrene
COPC - Chemical of potential concern
COPEC - Chemical of potential ecological concern
HI - Hazard index
NA - Not applicable
NFA - No further action

APPENDIX A

PHOTOGRAPHS OF SITES FTIR-32A AND FTIR-39

NTC, FORT IRWIN SITE FTIR-32A

NTC, FORT IRWIN SITE FTIR-39

APPENDIX B

TECHNICAL MEMORANDA

Jacklyn L Bowen
03/15/99 03:02 PM

To: Kristin A Shelton/User/Americas/Montgomery Watson@MW, Karen A Kramer/User/Americas/Montgomery Watson@MW
cc:

Subject: RE: FW: Goldstone lake Site/Mohave Ground Squirrel Habitat

---------- Forwarded by Jacklyn L Bowen/User/Americas/Montgomery Watson on 03/15/99 03:01 PM ----------

"Payton, Curtis SPK" <CPayton@spk.usace.army.mil> on 03/15/99 12:59:06 PM

To: "'Buzz Chernoff'" <BCHERNOF@ospr.dfg.ca.gov>, Susan Ellis <SELLIS@ospr.dfg.ca.gov>
cc: OPatrick@dtsc.ca.gov, gatestt@irwin.army.mil, Jacklyn L Bowen/User/Americas/Montgomery Watson@MW, "Payton, Curtis SPK" <CPayton@spk.usace.army.mil>, Bruce A Narloch/User/Americas/Montgomery Watson@MW
Subject: RE: FW: Goldstone lake Site/Mohave Ground Squirrel Habitat

---

In response to Buzz's note to Susan Ellis:
The reference is indeed to Site FTIR-39.

> -----Original Message-----
> From: Buzz Chernoff [SMTP:BCHERNOF@ospr.dfg.ca.gov]
> Sent: Monday, March 15, 1999 1:12 PM
> To: Susan Ellis; CPayton@spk.usace.army.mil
> Subject: Re: FW: Goldstone lake Site/Mohave Ground Squirrel Habitat
>
> Susan-
> I believe the biologist's report is in reference to Site 39 at Fort Irwin,
> the rocket test area on the lake bed. If so, this will provide the
> justification to remove this site from any further ecological
> consideration. Cheers . . . Buzz
>
> >>> "Payton, Curtis SPK" <CPayton@spk.usace.army.mil> 03/15/99 08:35AM >>>
> Per our meeting last Wednesday (March 10, 1999)
> /s/
> Curtis Payton
>
> > -----Original Message-----
> > From: Gates-Tull, Tiffany [SMTP:GatesTT@IRWIN.ARMY.MIL]
> > Sent: Monday, March 15, 1999 7:12 AM
> > To: 'Payton, Curtis'
> > Subject: FW: Goldstone lake Site/Mohave Ground Squirrel Habitat
> >
> > Please forward.
> > Tiffany S. Gates-Tull
> > Fort Irwin's Installation Restoration Program Manager
> > (760) 380-6713
> >
> >
> > From: Quillman, Mickey
> > Sent: Thursday, March 11, 1999 4:42 PM
> > To: Gates-Tull, Tiffany
> > Subject: Goldstone lake Site/Mohave Ground Squirrel Habitat
> >
> > Although the Mohave Ground Squirrel has been observed in the Goldstone

> > Deep Space Listening Station, the DIRP Site on Goldstone Lake is not
> > Mohave Ground Squirrel Habitat due to the fact that it is almost
> competely
> > devoid of vegetation. Mohave Ground Squirrel and its habitat should not
> > be a consideration during restoration of this site.
> >
> >
> > Mickey Quillman
> > NTC Biologist
> >

MEMORANDUM

MONTGOMERY WATSON

To:	Distribution	**Date:**	March 31, 1999
From:	Kristi Shelton	**Reference:**	Delivery Order 37
Subject:	Fort Irwin Site Visit		

A site visit was conducted at NTC Fort Irwin on March 23, 1999 to determine the potential plant tissue sampling locations recommended in the Workplan for Sites FTIR-32A, FTIR-38, FTIR-39, and FTIR-40. The attendees included: Tiffany Gates-Tull, Fort Irwin; John Christopher, Department of Toxic Substances Control (DTSC), Susan Ellis, Department of Fish and Game, Kevin, Department of Fish and Game, and Kristi Shelton, Montgomery Watson. The Fort Irwin biologist, Mickey Quillman, was unable to attend. Therefore, the plant species listed in the Workplan were not positively identified. However, sites with potential habitat were identified as areas containing substantial vegetation that might provide a food source for the Mojave ground squirrel.

The morning began with a visit to Site FTIR-38 Area 2. This site appeared to have adequate vegetation for the Mojave ground squirrel. In addition, numerous burrows were identified within the berms that could be potential ground squirrel habitat. This site was recommended for plant tissue sampling to determine the ecological risks to the Mojave ground squirrel.

The site visit at FTIR-40 Area 2 concluded that there was vegetation at the site. However, no previous surface soil samples have been collected at this site. The test pit near the septic tank area (the potential source of the contamination) was sampled in June 1997 at 5 and 10 feet below ground surface. Five soil borings are scheduled for this area and the possibility of collecting surface soil samples to determine the potential risk to the Mojave ground squirrel was discussed.

Site FTIR-40 Area 1.2 appeared to have Mojave ground squirrel burrows and vegetation. This site was recommended for plant tissue sampling to evaluate the ecological risks to the Mojave ground squirrel.

Site FTIR-39 was not visited since this site was previously eliminated from sampling. The final site visit was conducted at Site FTIR-32A (Lower Goat Mountain Landfill). This site was determined to have inadequate vegetation over the landfill area to be a possible habitat for the Mojave ground squirrel. In addition, no burrows or evidence of squirrel activity were discovered at the site. This site was not recommended for plant tissue sampling.

During the site visit there was discussion of the location of possible reference areas. The reference areas were decided on a per site basis. One reference area at each site was identified. These areas were selected based on similar plant species to the sites. The reference area for Site FTIR-38 Area 2 was identified as the area to the northwest of the site away from the berms. The Site FTIR-40 Area 2 reference area will be located south of the site on the other side of the road. The Site FTIR-40 Area 1,2 reference area will be located to the north of the site on the other side of the road.

Following the site visits the plant sampling methodology was discussed. Montgomery Watson will conduct the plant tissue sampling with the aid of a biologist subcontractor who will be able to identify all the plant species mentioned in the Workplan. The Department of Fish and Game will provide information on the parts of the plants that is the food source for the Mojave ground squirrel. These parts of the plants will be targeted for plant tissue sampling.

The site visit concluded with preliminary recommendations for plant tissue sampling at Sites FTIR-38 Area 2, FTIR-40 Area 1 2, and FTIR-40 Area 2. The Department of Fish and Game and DTSC will provide their final recommendations for plant tissue sampling locations following review of the Workplan.

Distribution:
Tiffany Gates-Tull (Fort Irwin - DPW)
John Christopher (DTSC)
Susan Ellis (Department of Fish and Game)
Curtis Payton (USACE)
Jacky Bowen (Montgomery Watson)
Karen Kramer (Montgomery Watson)
Bruce Narloch (Montgomery Watson)
Steve Crane (Montgomery Watson)

APPENDIX C

TOXICOLOGY PROFILES

TOXICOLOGY PROFILES

ARSENIC

(CAS RN 7440-38-2)

Arsenic is a naturally occurring element in the earth's crust usually found combined with one or more other elements. It is especially prevalent in ores that contain copper or lead. When combined with oxygen, chlorine, or sulfur it is referred to as inorganic arsenic, and when combined with carbon and hydrogen it is referred to as organic arsenic. This is an important distinction since inorganic forms are more prevalent in the environment than organic forms, and because inorganic forms are more toxic than organic forms. Both forms are released to the environment by human activities such as fossil fuel consumption, pesticide use and copper smelting, and via natural processes such as volcanic emissions, weathering of arsenic-containing minerals and ores, and forest fires (ATSDR, 1992). Soil usually contains about 5,000 µg/kg arsenic; food contains about 20 to 140 µg/kg; waste contains about 2 µg/L; and air contains about 0.02 to 0.10 $\mu g/m^3$.

Absorption/Distribution/Metabolism/Excretion

Water soluble inorganic arsenic compounds are substantially absorbed (more than 90 percent) from the GI tract of humans and laboratory animals. Estimates of absorption following oral exposure of humans to water soluble, inorganic arsenic compounds range from 54 to 95 percent (Bettley and O'Shea, 1975 cited in ATSDR, 1992). Less water soluble forms are absorbed to a smaller degree. For example, only 30 to 40 percent of an oral dose of arsenic trioxide, which has limited water solubility, is absorbed by laboratory animals (Marafante and Vahter, 1987 cited in ATSDR, 1992). Water soluble, inorganic arsenic compounds are also rapidly and substantially absorbed via inhalation in both animals and humans. Studies have indicated that 75 to 85 percent of deposited arsenic is absorbed from the lungs within 4 days (Holland et al., 1959 cited in ATSDR, 1992). Less soluble arsenic compounds (e.g., lead arsenate, gallium arsenide) are less

completely absorbed (about 55 percent of an administered dose absorbed in 3 days) (Marafante and Vahter, 1987 cited in ATSDR, 1992). There is little quantitative information on the dermal absorption of arsenic compounds in humans or in animals. However, one study has indicated that arsenic initially binds to the skin and is then slowly taken up into blood (Dutkiewicz, 1977 cited in ATSDR, 1992).

Once absorbed, arsenic is rapidly cleared from the blood in humans and animals (except in rats where it is bound to red blood cells) and distributed to the liver, kidney, lung, spleen, aorta, skin, hair and upper GI tract (Rhodes and Sanders, 1985; Liebscher and Smith, 1968 cited in ATSDR, 1992). These tissues are then cleared rapidly except for skin and hair, where arsenic tends to accumulate. Most absorbed arsenic is metabolized in the liver to methylated products and excreted in the urine (ATSDR, 1992). Since methyl derivatives of arsenic appear to be less toxic than inorganic arsenic, and since methylation results in less tissue retention of inorganic arsenic, methylation is viewed as a detoxification process.

Acute Toxicity

Oral doses of about 50 to 300 mg inorganic arsenic may be fatal to adults, and subchronic oral exposure to about 3 mg/day can be fatal to infants exposed via contaminated milk. On the basis of these observations, the minimum acute and subacute oral lethal dose in humans is estimated to be about 1 to 3 mg/kg/day (ATSDR, 1992). Inhalation or dermal exposure has not been associated with acute lethality in humans.

Acute or subacute oral exposures to high nonlethal, oral doses of arsenic ranging from about 0.04 to 3 mg/kg/day via drinking water or other types of ingestion (e.g., herbal preparation) produces a range of GI signs, with nausea, vomiting, and thirst being common. Very high oral doses may also produce acute encephalopathy (any degenerative disease of the brain), and injury to the liver consisting of necrosis (cell death) and fatty infiltration.

Animals are less sensitive to the toxic effects of arsenic than are humans. For example, reported lethal doses in animals (10 to 300 mg/kg) are significantly higher than lethal doses reported in humans (0.6 to 2 mg/kg).

Target Organ Toxicity

The EPA has published a chronic oral RfD of 3.0E-04 mg/kg-day for arsenic (IRIS, 2000). This value is based on observations of keratosis, hyperpigmentation, and possible vascular complications in humans exposed to arsenic in their drinking water. The purpose of the UF of 3 associated with this RfD is to account for the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty over whether the NOAEL of the critical study accounts for all senstive individuals. It should be noted that there was not a clear consensus among the Agency scientists on this RfD. risk managers should realize the flexibility afforded them in making regulatory decisions when uncertainty and lack of clear consensus are taken into account.

Developmental Toxicity

There is only suggestive evidence that exposure to arsenic results in developmental toxicity in humans (ATSDR, 1992). Studies in animals, however, do support the view that arsenic is a developmental toxicant, causing reduced birth weight, a variety of fetal malformations (both skeletal and soft tissue), and increased fetal mortality. These effects have been noted following inhalation exposure of mice (Nagymajtenyi et al. 1985), oral exposure of mice and hamsters (Baxley et al., 1981; Hood and Harrison, 1982; Hood et al., 1978 cited in ATSDR 1992), and intraperitoneal or intravenous injection of rats, mice, and hamsters (Beaudoin, 1974; Carpenter, 1987; Ferm and Carpenter, 1968; Ferm et al., 1971; Hanlong and Ferm, 1986c; Hood and Bishop, 1972; Mason et al., 1989; Willhite, 1981 cited in ATSDR, 1992). However, in all these cases the doses required to cause effects were high and often resulted in significant maternal toxicity or even lethality. It thus appears that although inorganic arsenic is a developmental toxicant, the developing fetus is not especially susceptible, and teratogenicity or fetotoxicity are

unlikely to be of concern except at doses that are also toxic to the pregnant female (ATSDR, 1992).

Genotoxicity

The results of tests for genotoxicity of arsenic are mixed, but in general the inorganic arsenicals appear to be either inactive or weak mutagens (Jacobson-Kram and Montalbano, 1985 cited in ATSDR, 1992) that are able to produce chromosomal effects (aberrations, sister chromatid exchange) in most systems. Studies of humans have detected higher than average incidence of chromosomal aberrations in peripheral lymphocytes, both after inhalation exposure (Beckman et al., 1977; Nordenson et al., 1978 cited in ATSDR, 1992) and oral exposure (Burgdorf et al., 1977; Nordenson et al. 1979 cited in ATSDR, 1992).

Cardiotoxicity. Chronic oral exposure to arsenic is also associated with cardiological and vascular effects. Injury and abnormal function of cardiac tissue have been reported in children ingesting water containing as little as 0.6 to 0.8 mg/L arsenic. Peripheral vascular disease leading to gangrene of the toes and feet ("Blackfoot disease") has been reported in a Taiwanese population ingesting drinking water containing 0.4 to 0.6 mg/L arsenic (Chen et al., 1988b; Chi and Blackwell, 1968; Tseng et al., 1968 cited in ATSDR, 1992). Similar peripheral vascular lesions have been reported in vintners exposed to arsenical pesticides, in persons in Chili consuming water containing about 0.8 mg/L arsenic, and in patients from a region in Mexico where arsenic toxicity is endemic (ATSDR, 1992). (It should be noted that some investigators have questioned the role of arsenic in Blackfoot disease and have suggested that the causative factor may be a fluorescent arsenic-containing compound of unknown structure which is present in water where "Blackfoot disease" is endemic.)

Carcinogenicity

There is evidence that chronic oral exposure to elevated levels of arsenic increases the risk of skin cancer in humans. The largest study of arsenic-induced skin cancer has been described by

Tseng (1968 cited in ATSDR, 1992)). This study focused on a large population in Taiwan where arsenic levels in deep wells used for drinking water ranged from 0.001 to 1.82 mg/L, with average levels of around 0.4 to 0.6 mg/L. Based on examination of over 40,000 exposed people the excess skin cancer rate was found to be 10.6/1000 (or about 1×10^{-2}). On the basis of these and other (Wv et al., 1989; Zaldiver, 1974 cited in ATSDR, 1992) the EPA cautions that the design of this study limits its usefulness in risk estimation. Arsenic-induced skin cancer has also been attributed to water supplies in Chile, Argentina and Mexico (Borgono and Greiber, 1972; Bergoglio, 1964; Cebrian et al 1983 cited in ATSDR, 1992).

The EPA has concluded that the Tseng (1977) study is the most appropriate basis for the derivation of an oral unit risk and slope factor for arsenic, although because of its study design considerable uncertainty in dose response should be recognized. Based on this study, a unit risk of 5E-05 $(ug/L)^{-1}$ has been published (IRIS, 2000) The associated slope factor is calculated to be 1.5E+00 $(mg/kg\text{-}day)^{-1}$. IRIS (2000) notes that a recent memorandum by the Administrator of the EPA, while recommending that the above slope factor be adopted, also notes that the "uncertainties associated with ingested inorganic arsenic are such that estimates could be modified downwards as much as an order of magnitude, relative to risk estimates associated with most other carcinogens. In such instances, the management document must clearly articulate this fact and state the factors that influenced such a decision." (This seems to imply that a unit risk of 5.0E-06 for arsenic may be appropriate, and that an associated slope factor of 1.5E-01 $(mg/kg\text{-}day)^{-1}$ may be acceptable)

The ATSDR toxicological profile for arsenic notes that several types of internal tumors have also been associated with oral arsenic exposure (ATSDR, 1992). For example, hepatic angiosarcoma, a rare tumor in the general population, occurs at increased frequency in persons exposed to Fowler's solution (potassium arsenite), or to arsenical pesticides. Also, exposure to arsenic through ingestion of water containing 0.4 to 1.1 mg/L arsenic has been associated with increased occurrence of bladder, lung and liver cancer. Limited observations suggest arsenic might be associated with several other types of cancer as well, including cancer of the mammary gland, cancer of the lymphatic tissues, leukemia, and renal adenocarcinoma (Kasper et al., 1984; Lender

et al., 1975; Regelson et al., 1968, Roth, 1957; Sommers and McManus, 1953; Chen et al., 1985, 1986, 1988a, 1988b, 1988c; Chiang et al., 1988; Wu et al., 1989 cited in ATSDR, 1992).

Chronic Toxicity

Chronic exposure to arsenic via inhalation results in an elevated risk of lung cancer in humans. Studies of smelter workers in the United States, Sweden, and Japan have all found an association between occupational arsenic exposure and lung cancer mortality (Enterline and Marsh, 1982; Lee-Feldstein, 1983; Axelson et al., 1978; Tokudome and Kuratsune, 1976; Rencher et al., 1977 cited in ATSDR, 1992). Studies of pesticide manufacturing workers, of arsenical pesticide applicators, and of a population located near a pesticide manufacturing plant have also revealed an excess of lung cancer deaths among exposed persons (Ott et al., 1974; Mabuchi et al., 1979; Matanoski et al., 1981; Roth, 1958, as cited in ATSDR, 1992).

The EPA has derived geometric means of unit risks from different data sets obtained from workers occupationally exposed at two different smelters in the United States. The final estimate of an inhalation unit risk is the geometric mean of these two values and is 4.3E-03†$(\mu g/m^3)^{-1}$ from which an inhalation slope factor of 1.5E+01 $(mg/kg/day)^{-1}$ has been derived (IRIS, 2000).

It is interesting to note that in laboratory animals oral exposure to arsenic does not result in cancer; and, there is only suggestive evidence that exposure to arsenic via inhalation induces lung cancer.

REFERENCES

Agency for Toxic Substances and Disease Registry. Draft, 1992. Toxicological Profile for Arsenic. Update.

U.S. Environmental Protection Agency (EPA). Integrated Risk Information System (IRIS). 2000.

BARIUM

(CAS RN 7440-39-3)

Barium is a yellowish-white, slightly lustrous metal with a body-centered cubic structure (Merck, 1983, as cited in HSDB, 1995) It is used in paints, soap, paper, and rubber, and in the manufacture of ceramics and glass (Doull, et al., 1986, as cited in HSDB, 1995). It is also used as a component in various proprietary nodularizing and deoxidizing alloys, in the manufacture of alloys for such products as nickel barium parts used in ignition equipment for automobiles, and in the manufacture of lithopone and television picture tubes (Venugopal, et al., 1978, as cited in HSDB, 1995). Barium is soluble in alcohol but insoluble in benzene; it has a boiling point of 1640°C and a melting point of 725°C (Weast, 1989, as cited in HSDB, 1995). Naturally occurring barium is a mixture of seven stable isotopes; thirteen other radioactive isotopes are known to exist (Weast, 1989, as cited in HSDB, 1995).

Absorption/Distribution/Metabolism/Excretion

Absorption of barium following inhalation exposure by humans has not been widely studied; however, results of studies with experimental animals suggest that the rate and extent of absorption of barium from the respiratory tract depends on the exposure level, and the solubility of the particular form of barium that was administered (ATSDR, 1991). As with other metals, barium is probably very poorly absorbed from the GI tract; the International Commission for Radiation Protection (ICRP) estimates that the GI absorption of barium is less than 5% (ICRP, 1973, as cited in ATSDR, 1991). Barium is not lexpected to cross intact skin because of the high polarity of the forms in which it is most commonly encountered (ATSDR, 1991); therefore, dermal absorption of barium is not likely.

Studies in humans indicate that barium distributes predominantly to the skeleton and teeth. The route of exposure is presumed to be mostly oral (ATSDR, 1991). Barium occurs mostly in the bones and teeth of humans. Very little is found in blood plasma or soft tissues; but, when it is

detected in the organs, it is found in the eyes, lungs, skin, and adipose tissue in humans (Schroeder et al., 1972, as cited in ATSDR, 1991).

Since barium is an element, it is not metabolized in the body, but it may be metabolically transported or incorporated into complexes or tissues (ATSDR, 1991).

Barium taken by mouth is poorly absorbed, therefore, most of the dose is excreted in the feces (Tipton et al., 1966, as cited in ATSDR, 1991). Dogs that received barium by gavage excreted most of the dose within a few days and less then 3% of the initial body burden remained in the body after two weeks (Cuddihy and Griffith, 1972, as cited in ATSDR, 1991).

Acute Toxicity

No studies were available regarding death in humans or animals after inhalation or dermal exposure to barium. However, mortality has been reported to occur in a number of cases where humans have been acutely exposed to barium through accidental or intentional ingestion (ATSDR, 1991). These observations are supported by findings from acute studies with rodents that indicate barium is toxic by the oral route (Borzelleca et al., 1988, as cited in ATSDR, 1991). Reduced life span also has been observed in chronic oral studies with mice. These results from human case studies and acute and chronic studies with rodents suggest that humans who are exposed orally to high levels of barium may be at increased risk for mortality (ATSDR, 1991).

Target Organ Toxicity

Barium has been associated with a number of adverse health effects in both humans and experimental animals. Both human and animal evidence suggests that the cardiovascular system may be one of the primary targets of barium toxicity (ATSDR, 1991).

The US EPA has calculated an oral RfD of 7.0E-02 mg/kg/day based on increased blood pressure observed in subchronic to chronic human drinking water studies (IRIS, 2000). The UF of 3 is used to account for sensitive individuals.

Respiratory Effects. Benign pneumoconiosis has been observed in workers exposed occupationally by inhalation to barium (Doig et al., 1976, as cited in ATSDR, 1991). Acute intravasation of barium sulfate into the circulatory system of an adult female following a barium enema caused the compound to be deposited in blood cells throughout the body, including the lungs, and resulted in respiratory failure (Cove et al , 1974, as cited in ATSDR, 1991).

Developmental Toxicity

Results of a study which evaluated the correlation between barium concentrations in drinking water and human congenital malformation rates of the CNS indicated there was no correlation. However, another study suggested that exposure to barium may potentially be associated with adverse developmental effects. Reduced survival, underdevelopment, lowered body weight, decreased ability of the peripheral nervous system, and various blood disorders were reportedly noted in the offspring of rats following inhalation to barium for an intermediate exposure period. In the same study, increased mortality, increased leukocyte count, disturbances in liver function, and increased urinary excretion of hippuric acid were noted in the offspring of rats treated orally to barium during conception and pregnancy (Tarasenko et al., 1977, as cited in ATSDR, 1991).

Reproductive Toxicity

There are no studies regarding the reproductive effects of barium in humans (ATSDR, 1991). However, two animal studies have provided limited information suggesting that humans exposed to barium may be at increased risk for developing reproductive effects. Decreased ovary weight and decreased ovary/brain weight ratio have been noted in rats following acute oral exposure to barium. Intermediate inhalation exposure to barium has been associated in rats in one limited study with disturbances in spermatogenesis, shortened estrous cycle, and alterations in the

morphological structure of the ovaries and testes (Tarasenko et al., 1980, as cited in ATSDR, 1991).

Genotoxicity

Available data relating to the genotoxic effects of barium are derived from in vitro studies only; there were no available data regarding the genotoxicity of barium in vivo. A single recombination assay in which Bacillus subtilis was exposed to barium was negative for mutagenicity. Results of a test evaluating the fidelity of DNA synthesis in an avian myeloblastosis virus DNA polymerase system indicate that barium did not affect the accuracy of DNA replication (Nishioka, 1975 as cited in ATSDR, 1991).

Carcinogenicity

Two chronic oral studies which examined the incidence of tumors in rats and mice exposed to barium acetate in drinking water for lifetime were negative for carcinogenicity; however, they were inadequate for evaluating carcinogenic effects because insufficient numbers of animals were used, a complete histological examination was not performed, and only one exposure dose was evaluated (Schroeder and Mitchener 1975, as cited in ATSDR, 1991). Precancerous lesions were reported in one study in which a woman was treated on the cervix with a barium chloride solution; however, the relevance of this limited observation cannot be determined because only one subject was treated and because the vehicle solution was not specified (Ayre, 1966, as cited in ATSDR, 1991). Barium has not been evaluated by EPA for human carcinogenic potential (IRIS, 1995).

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CADMIUM

(CAS RN 7440-43-9)

Cadmium occurs naturally in the earth's crust, and low levels of cadmium are found in most waters. It is most often encountered in combination with other elements such as oxygen, chlorine, or sulfur. The largest anthropogenic source of cadmium in the general environment is the burning of fossil fuels (such as coal or oil) or the incineration of municipal waste materials. It may also escape into the air from zinc, lead, or copper smelters. Smoke from cigarettes also contains cadmium and most people who smoke have about twice as much cadmium in their bodies as nonsmokers.

Absorption/Distribution/Metabolism/Excretion

Cadmium is poorly absorbed from the GI tract. An average of only 3 to 6 percent of an oral dose of cadmium chloride has been reported to be retained by humans, with individual values ranging from about 1 to†7 percent. In animals, 0.5 to about 3 percent of an oral dose is retained.

Absorption via inhalation is considerably greater than via ingestion, although this is limited to cadmium adsorbed to particulates of 0.1 mm or smaller which tend to be deposited in alveoli. For example, about 60 to 70 percent of a dose of cadmium instilled in rat lungs has been reported to be absorbed. No direct measurements of alveolar absorption in humans are available, but calculations based on the increased body burden in smokers compared to that in nonsmokers suggest that respiratory absorption in man is probably about 30 to 60 percent. Small quantities of cadmium may also be absorbed through the skin.

Once absorbed, cadmium is widely distributed in the body with the major portion going to the liver and to the renal cortex of the kidney, where it accumulates. Cadmium is not known to undergo any direct metabolic conversions. It does, however, induce increased synthesis of the protein metallothionen, to which it is bound. When present in plasma the cadmium-metallothionen complex is readily diffusible and filterable at the glomerulus and is effectively

reabsorbed from the glomerular filtrate. This process effectively prevents efficient excretion of cadmium in the urine and contributes to the accumulation of cadmium in the kidney and liver.

Acute Toxicity

Acute oral exposure to 1,500 to 8,900 mg (20 to 130 mg/kg) cadmium or acute inhalation exposure to 40 to 50 mg/m^3 cadmium for 1 hour or to 9 mg/m^3 cadmium for 5 hours has been reported to cause death in humans. High oral exposures result in severe irritation of the GI tract including vomiting, diarrhea and abdominal pain. The concentration of cadmium in water that will induce vomiting is approximately 15 mg/L. High inhalation exposures are intensely irritating to respiratory tissues. Typical acute respiratory effects include severe tracheobronchitis (inflammation of the trachea and bronchi), pneumonitis (inflammation of the lung), and pulmonary edema (retention of fluid in the lung).

Acute oral LD_{50} values for cadmium in laboratory animals range from 50 to 350 mg/kg. Acute inhalation LD_{50} values range from 500 to 15,000 mg/m^3-minute.

Acute parenteral exposure of male rats to 2.2 mg/kg cadmium has resulted in marked necrosis and atrophy of the testes. Very large acute oral doses (100 mg/kg) have also been reported to cause similar testicular damage. Acute injections of cadmium also result in massive ovarian hemorrhage in female rats given subcutaneous injections of 2.3 to 4.6 mg/kg. Similar effects have been observed in mice. Hemorrhage and necrosis of the placenta of rats has also been observed following subcutaneous injection of 4.5 mg/kg cadmium on days 17 to 21 of gestation.

Target Organ Toxicity

The most commonly observed effects associated with chronic cadmium exposure are related to the accumulation of cadmium in the kidney, which occurs regardless of the route of exposure. It has been estimated that when accumulation of cadmium in the renal cortex reaches 200 µg/L wet weight renal cortex, the types of renal dysfunction described below will result.

Cadmium is unusual in relation to most of the substances for which an oral RfD has been determined in that a vast quantity of both human and animal toxicity data are available. The RfD is based on the highest level of cadmium in the human renal cortex not associated with significant proteinuria, which is 200 µg cadmium/gm wet weight human renal cortex. A toxicokinetic model was used to determine the level of chronic oral exposure that would result in this level of cadmium accumulation in the kidney (EPA, 1985 cited in ATSDR, 1989). Assuming that 2.5 percent of cadmium in food and 5 percent of cadmium in water is absorbed, and that 0.01 percent of the body burden of cadmium is eliminated each day, the model estimates that a daily dose of 0.005 mg/kg-day cadmium in water, or 0.01 mg/kg-day cadmium in food would result in a concentration of 200 µg Cd/gm wet weight renal cortex. These values are thus considered No Observed Adverse Effect Levels (NOAELs). When these values are adjusted to account for the possibility that some humans may be especially sensitive to cadmium-induced renal disease they become 0.0005 mg/kg-day for water and 0.001 mg/kg-day for food.

The EPA has derived an oral RfD of 0.0005 mg/kg-day for cadmium in water and 0.001 mg/kg-day for cadmium in food based on the occurrence of proteinuria in humans (IRIS, 2000). EPA has not yet derived an inhalation RfC or RfD.

Renal Toxicity. Proximal tubular damage in the kidney as indicated by the presence of $beta_2$-microglobulin (microglobulinuria), lysozyme, ribonuclease, immunoglobulin light chains and retinol-binding protein in the urine occurs in workers exposed to cadmium via inhalation. This may be accompanied by depressed tubular reabsorption of other solutes, as well, such as enzymes, amino acids, glucose, calcium and inorganic phosphate. Microglobulinuria has also been reported in males in cadmium-polluted areas of Japan and Belgium.

These effects typically occur only after relatively long exposures to cadmium, or if cadmium levels have been acutely elevated. Following acute exposures, the relatively slow development of toxicity may represent the time required for cadmium to be redistributed in the body and the time required to reach a critical level in the renal cortex. Several estimates have indicated that

chronic exposures to 200 to 352 µg/day (about 0 003 to 0 005 mg/kg-day) cadmium over a period of 45 to 50 years would result in a critical level of cadmium in the renal cortex, and subsequent tubular proteinuria in at least 10 percent of an exposed population A dose of 50 µg/day (about 0 0007 mg/kg-day) was estimated to cause tubular proteinuria in about 1 percent of an exposed population after 45 years. Via inhalation, it has been estimated that 10 percent of a worker population exposed to a concentration of 50 mg/m^3 would develop proteinuria in 10 years and that 1 percent would be affected at an exposure level of 16 $\mu g/m^3$. Exposures to 2 2 and 0 7 $\mu g/m^3$ cadmium for 50 years would result in similar renal dysfunction in 10 percent and 1 percent of the exposed population, respectively.

The renal response to cadmium exposure in laboratory animals appears to be similar to humans. In cadmium exposed rabbits, enzymuria occurred when the cadmium level in renal cortex reached 117 µg/L (dose not provided); aminoaciduria was observed at about 200†µg/L; and, low molecular weight proteinuria occurred when the renal cortex cadmium concentration was about 300 µg/L.

Musculo-Sketetal Toxicity. Painful bone disorders, including osteomalacia (softening of the bones), osteoporosis (bone thinning) and spontaneous bone fracture, have been observed in some humans chronically exposed to high levels of cadmium. These symptoms have been most thoroughly studied in postmenopausal women living in a cadmium-contaminated area in Japan where the affliction is called Itai-Itai disease. Osteomalacia has also been observed in some occupationally exposed workers. These effects on bone are generally considered to be indirect, arising as a consequence of cadmium-induced renal disease. (It should be noted that the role of cadmium as a direct cause of Itai-Itai disease is disputed. Some investigators argue that a nutritional deficiency plays the most important role and that cadmium exposure may simply enhance the incidence or severity of the disease through its effects on the kidney.)

Hematotoxicity. Some other effects reported to be associated with chronic occupational cadmium exposure are lowered hemoglobin concentrations and decreased packed cell volumes, and malabsorption of iron and various other solutes. Dyspnea (difficult breathing), emphysema,

decreased forced vital capacity, and increased mortality from respiratory disease have also been reported among populations chronically exposed to cadmium via inhalation. A positive correlation between increased mean renal cadmium concentrations in humans and the incidence of death from hypertensive disease has been reported by several laboratories. However, the incidence of hypertension is not known to be increased in human populations known to have been exposed to cadmium. A link between cadmium and hypertensive disease is therefore not certain.

Long-term oral cadmium administration has also been associated with anemia in mice and rats, and with increased passive avoidance behavior and neurobehavioral changes in rats.

Hepatotoxicity. Chronic oral exposure of rabbits to about 13 µg/kg/day cadmium in their drinking water, and of rats to 17 mg/L cadmium has been associated with structural changes in the liver. Changes in carbohydrate and ATP metabolism in the livers of rats have been reported following exposure to 25 ppm cadmium in their food.

Cardiotoxicity. A number of studies indicate that function of both the heart and the vascular system of laboratory animals may be impaired by cadmium exposure. Characteristic effects in the heart include decreased speed and strength of contractions. The effects in vascular smooth muscle may be either vasodilation or vasoconstriction, depending on conditions. Chronic exposure of rats to about 0.5 mg/kg-day cadmium in their drinking water resulted in small but significant increases in average systolic blood pressure. Changes were also observed in electrocardiograms of exposed rats. This hypertensive effect appears to be biphasic, reaching a maximum effect at doses around 0.1 mg/kg-day, but having no effect at exposure levels of 5 to 10† mg/kg-day.

Respiratory System Toxicity. Emphysema in rabbits and pulmonary fibrotic lesions in rats have been observed following chronic inhalation exposure to cadmium. Decreased calcium content in bone accompanied by marked rental tubular injury and increased urinary excretion of calcium have also been reported to follow chronic cadmium exposure of rats via inhalation.

Immunotoxicity. Relatively low doses of cadmium have been reported to alter the immune response in experimental animals. For example, decreased levels of spleen plaque-forming cells have been observed in mice exposed to 0.6 mg/kg-day for 10 weeks. Mice exposed to 5 to 50 mg/L cadmium in drinking water for 3 weeks exhibited immune suppression. Cadmium concentrations in the renal cortex of affected mice in these experiments was only 0.3 to 6.0†µg/L, which is considerably lower than the suggested critical level of cadmium in human renal cortex (200 µg/L) associated with toxicity.

Developmental and Reproductive Toxicity

As noted above acute parenteral exposure of rats to 2.2 mg/g or acute exposure to high oral doses, results in marked necrosis and atrophy of the testes. Single injections of 2.3 to 4.3 mg/kg cadmium to female rats or mice causes massive ovarian hemorrhage. Other reproductive effects include severely depressed testosterone levels and no viable sperm in male rats injected with doses of 1.8 mg/kg of cadmium chloride 14 days previously. Similarly, a marked reduction in sperm count of male rats injected with 1 mg/kg of cadmium chloride 15 days previously has been reported, although no decrease in fertility was observed until the dose reached 5 mg/kg.

Single injections (intravenous or subcutaneous) of high doses of cadmium during gestation result in teratogenicity and increased fetal mortality in hamsters (effective dose: 2 mg/kg) and mice (effective dose: 0.63 mg/kg). When exposure is via ingestion (about 10 to 15 mg/kg-day) the most common finding is decreased weight of offspring, usually without significant teratogenic or developmental effects. Congenital tail malformation was observed in a three generation study of rats exposed to 10 mg/L of cadmium chloride in drinking water (about 1 mg/kg-day) and sirenomelia (fused legs) was observed in offspring of rats dosed by gavage with cadmium chloride (40 mg/kg) on days 7 to 16 of gestation. Impaired neurobehavioral development in rats has been reported following exposure of female rats via gavage (0.4 or 4.0 mg/kg, given for 5 weeks preceding and continuing through gestation), via drinking water (60 mg/L given during

gestation), or via inhalation (0.02 or 0.16 mg/m^3, for 5 months preceding and continuing through gestation.

Exposure to airborne concentrations of 0.2 to 0.6 mg/m^3 during gestation results in a reduction of both maternal and fetal weight gain but no significant teratogenicity. Similarly, decreased pup weight and elevated perinatal mortality was observed in rats exposed to cadmium by inhalation to 3 mg/m^3, but no teratogenic or developmental effects were noted.

It should be noted that cadmium has not been observed to cause teratogenic or other developmental or reproductive effects in exposed humans.

Genotoxicity

Available data suggest that cadmium is mutagenic in mammalian cell culture assay systems. It is also mutagenic in both the mouse lymphoma assay and the Chinese hamster cell assay.

Conflicting results have been reported for chromosomal aberration studies using human lymphocytes from exposed workers, and using human and animal cell lines treated with cadmium in vitro. Chromosomal aberrations have been reported in hamster cells treated with cadmium sulfate or cadmium chloride and in human lymphocytes treated with cadmium acetate and cadmium sulfide. However, human lymphocytes treated with cadmium chloride in vitro showed no chromosomal aberrations. Lymphocytes taken from humans with above average cadmium exposure have produced both positive and negative responses.

Carcinogenicity

Epidemiological studies in humans exposed to cadmium provide limited evidence that inhaled cadmium is a human lung carcinogen. Thun et al. (1985 cited in ATSDR, 1989) reported that mortality from respiratory cancer tended to increase in a dose-dependent fashion in workers with cumulative levels of less than 585, between 585 -2,920 and greater than 2,920 mg cadmium/m^3-

days, although the increase was statistically significant only in the high dose group These exposures correspond to time-weighted daily average levels of 168, 727 and 2,522†$\mu g/m^3$ cadmium, respectively. This observation is complicated by the fact that workers may have been exposed to other carcinogenic chemicals, including arsenic; and, exposure levels to both cadmium and arsenic were higher in previous years than in more recent years. Although some researchers have concluded that the observed excess in lung cancer is more likely to be due to arsenic than cadmium exposure (White 1985, Lamm 1987 cited in ATSDR, 1989), Thun et al. (1985) considered possible confounding by arsenic and concluded that arsenic exposure could not explain the observed excess in lung cancer.

An excess lung cancer risk has also been observed in three other studies but the EPA has concluded interpretation of the results of these studies is compromised by the presence of other carcinogens (arsenic, smoking) in the exposure, or by a small study population (Varner 1983; Sorahan and Waterhouse 1983; Armstrong and Kazantzis, 1983 cited in ATSDR, 1989).

Four studies of workers exposed to cadmium dust or fumes have provided evidence of a statistically significant association with prostate cancer (Kipling and Waterhouse, 1967; Lemen et al., 1976; Holden, 1980; Sorahan and Waterhouse, 1983 cited in ATSDR, 1989), but the total number of cases was small in each study.

Studies in animals have demonstrated that chronic inhalation exposure to cadmium chloride is associated with increased frequency of lung tumors. Takenaka et al. (1983 cited in ATSDR, 1989) exposed rats to aerosols of cadmium chloride at concentrations of 0, 12.5, 25 or 50†$\mu g/m^3$ for 18 months and observed a dose related increase in primary lung carcinoma frequency of 0, 15, 53 and 71 percent respectively. Mammary tumors in female rats, and tumors at multiple sites in male rats have been reported to follow intrathecal instillation of cadmium oxide (Sanders and Mahaffey, 1984 cited in ATSDR, 1989).

EPA has calculated unit risk values of lung cancer associated with lifetime exposure to a concentration of 1 $\mu g/m^3$ from both animal data (Takenaka et al., 1983 cited in ATSDR, 1989)

and human data (Thun et al., 1985 cited in ATSDR, 1989). The resulting values are 9.2×10^{-2} and 1.8×10^{-3} respectively. The California EPA (Cal EPA) has established an inhalation slope factor of 15 (Cal EPA, 1992). A unit risk of 1.8×10^{-3} $(ug/m^3)^{-1}$ has also been published (IRIS, 2000).

There is no evidence to suggest cadmium is carcinogenic to either animals or humans via oral exposure

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COBALT

(CAS RN 7440-48-4)

Cobalt is essential as a component of vitamin B_{12} required for the production of red blood cells and prevention of pernicious anemia. Cobalt is a relatively rare metal produced as a byproduct or coproduct of the refining of other mined metals such as copper and nickel. It is used in high-temperature alloys and in permanent magnets. Its salts are useful in paint driers, as catalysts, and in the production of numerous pigments (Goyer, 1986)

Inhalation is a potential route of exposure occurring most commonly when material containing cobalt powders are handled manually, or where cobalt materials are subjected to grinding. Dermal exposure to cobalt can occur in any operation where these materials are handled manually, or where solutions containing cobalt are present.

Absorption/Distribution/Metabolism/Excretion

In a study using radioactive cobalt administered orally or intravenously it was found that cobalt does not accumulate in tissues, suggesting that absorption and retention is poor (Browning, 1969, as cited in HSDB, 1994). Cobalt is stored in intestinal mucosa and subsequently lost through normal desquamation of epithelium (Venugopal, 1978, as cited in HSDB, 1994).

Acute Toxicity

Acute, high oral or parenteral doses of cobalt in humans and animals induced myocardial degeneration, often leading to mortality, erythropoiesis, enlarged thyroid, and in animals, renal tubular degeneration (Elinder and Friberg, 1986). Chronic ingestion from the consumption of beer containing high concentrations of cobalt has been associated with a condition called "beer-drinkers cardiomyopathy," which includes polycythemia, goiter, and marked myocardial degeneration and mortality. The therapeutic use of 0.16 to 0.32 mg Co/kg/day in anemic

anephric dialysis patients for 12 to 32 weeks induced a significant but reversible rise in blood hemoglobin (HEAST, 1992).

Occupational (inhalation and dermal) exposure has been associated with allergic dermatitis, chronic interstitial pneumonitis, reversibly impaired lung function, occupational asthma, and myocardial effects (Goyer, 1986). Cobalt has been determined to be the etiologic factor in hard metal disease, the syndrome of respiratory symptoms and pneumoconiosis associated with inhalation exposure to dusts containing tungsten carbide with cobalt powder as a binder (Elinder and Friberg, 1986). The lowest occupational air concentration of cobalt associated with hard metal disease was 0.003 Co/m^3 (Sprince et al., 1988). It should be noted that the workers were also exposed to tungsten and sometimes to titanium, tantalum, and niobium (Elinder and Friberg, 1986). Similar lung effects have been seen in animals exposed to cobalt by inhalation.

Target Organ Toxicity

Important target organs in orally exposed humans are the heart, erythrocyte, and thyroid. Target organs for occupational exposure are the skin, lungs, and heart.

Carcinogenicity

No studies were located regarding the carcinogenicity of cobalt in humans.

Single and repeated subcutaneous or intramuscular injection of cobalt powder and salts to rats may cause sarcomas at the site of injection (Gilman, 1962; as cited in Casarett and Doull, 1986; Venupogal, 1978; Friberg 1986; Seiler 1988; as cited in HSDB, 1994). It must be noted that there is little evidence of carcinogenicity from any other route of exposure.

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COPPER

(CAS RN 7440-50-8)

Copper is a reddish metal that occurs naturally in rock, soil, water, sediment, and air. Its average concentration in the earth's crust is about 50 parts per million. It is insoluble in hot and cold water but soluble in nitric acid or hot sulfuric acid (Weast, 1983, as cited in HSDB, 1995). Copper also occurs naturally in plants and animals. It is an essential element for all known living organisms including humans and other animals (ATSDR, 1990). Copper can be easily molded or shaped. Its reddish color is most commonly seen in the United States penny, electrical wiring, and some water pipes (ATSDR, 1990). Copper is used as a metal for electrical and electronic products (e.g., wire), building construction (e.g., plumbing pipes), industrial machinery, and equipment. In the transportation industry it is used in automobiles (HSDB, 1995). Copper has a boiling point of 2595°C, and a melting point of 1083°C (The Merck Index, 1983, as cited in HSDB, 1995).

Absorption/Distribution/Metabolism/Excretion

Copper oxide was observed in alveolar capillaries three hours after rats were exposed to a welding dust aerosol generated from pure copper wires (Batsura, 1969, as cited in ATSDR, 1990). Copper is absorbed in the stomach and small intestine. The site of maximal copper absorption is not known for humans, but it is assumed to be the stomach and upper intestine because of the rapid appearance of ^{64}Cu in the plasma after oral administration (Bearn and Kunkel, 1955, as cited in ATSDR, 1990).

Copper is absorbed from the GI tract as ionic copper, or is bound to amino acids (Crampton et al., 1965 as cited in ATSDR, 1990). Numerous factors may affect copper absorption. These factors include: (1) competition with other metals, including zinc and cadmium (Davies and Campbell, 1977, as cited in ATSDR, 1990); (2) the amount of copper in the stomach (Farrer and Mistilis, 1967, as cited in ATSDR, 1990); (3) certain dietary components; and (4) form of copper.

The absorption of copper appears to be inversely related to the amount of copper in the GI tract (Strickland et al., 1972, as cited in ATSDR, 1990)

Absorbed copper loosely binds to plasma albumin and amino acids in the portal blood, and is taken to the liver (Marceau et al., 1970, as cited in ATSDR, 1990). In the liver, copper is incorporated into ceruloplasmin and released into the plasma. Radioactive copper does not accumulate in extrahepatic organs until after the emergence of ceruloplasmin-^{64}Cu, suggesting that ceruloplasmin is a copper donor for the tissues (Owen, 1965, as cited in ATSDR, 1990).

The metabolism of copper consists mainly of its transfer to and from various organic ligands, most notably sulfhydryls and imidazole groups on amino acids and proteins. Several specific binding proteins for copper have been identified that are important in the uptake, storage, and release of copper from tissues. In the liver and other tissues, copper is stored bound to metallothionein and amino acids and in association with copper-dependent enzymes. Several studies have shown that copper exposure induces metallothionein synthesis (Mercer et al., 1981, as cited in ATSDR, 1990).

Bile is the major pathway for the excretion of copper. After the oral administration of radioactive copper as copper acetate in healthy humans, 72% was excreted in the feces (Bush et al., 1955, as cited in ATSDR, 1990). Normally, 0.5 - 3.0 percent of daily copper intake is excreted into the urine (Cartwright and Wintrobe, 1964, as cited in ATSDR, 1990). Biliary excretion of copper does not increase proportionally with dosage, suggesting that the hepatobiliary transport of copper is saturable (Gregus and Klaassen, 1986, as cited in ATSDR, 1990). Thus, at high copper intakes, urinary copper excretion increases (Gitlan et al., 1960, as cited in ATSDR, 1990).

Acute Toxicity

The only significant example of copper toxicity in humans is Wilsonís disease (Hepatolenticular degeneration), an autosomal recessive disorder that affects normal copper homeostasis. The disease is characterized by excessive retention of hepatic copper, decreased concentration of

plasma ceruloplasmin, impaired biliary copper excretion, and hypercupruria The systemic manifestations of Wilsonís disease are hepatic and renal lesions and hemolytic anemia (Schroeder et al , 1966, as cited in ATSDR, 1990).

There are several reports of humans dying as a result of copper poisoning. Most of these involve the ingestion of large amounts of copper. Chuttani et al. (1965, as cited in ATSDR, 1990) attributed these deaths to extensive hepatic centrilobular necrosis. Deaths in animals given >250 mg Cu/kg/day in the diet have also been attributed to extensive hepatic centrilobular necrosis.

Target Organ Toxicity

The primary toxicological effects of consuming high levels of copper in humans is GI irritation, manifested as vomiting, nausea, diarrhea, and anorexia. Centrilobular necrosis of the liver and necrosis and sloughing of tubular cells in the kidney have been observed in individuals dying from copper poisoning (Chuttani et al , 1965, as cited in ATSDR, 1990).

The US EPA has listed a drinking water standard for copper of 1.3 mg/L (HEAST, 1997) from which an oral RfD of 3.7E-2 mg/kg/day can be derived.

Immunological Effects. Dermal exposure to copper results in contact allergic dermatitis in some individuals (Barranco, 1972, as cited in ATSDR, 1990). Inhalation studies in mice confirm the finding of impaired immune function after exposure to copper (Drummond et al., 1986, as cited in ATSDR, 1990).

Neurological Effects. Neurological effects have not been observed in healthy humans exposed to high levels of copper in drinking water. However, a clinical manifestation of Wilsonís disease is CNS degenerative changes. Symptoms include poor coordination, psychological impairment, tremor, disturbed gait, and rigidity (Strickland and Leu, 1975, as cited in ATSDR, 1990). Although neurotoxicity has not been observed in animals exposed to high levels of copper,

increased concentration of copper in the brain has been observed in rats given high levels of copper orally or intraperitoneally (DeVries et al., 1986, as cited in ATSDR, 1990).

Developmental Toxicity

Increased fetal mortality and developmental abnormalities have been observed in mice, mink, and hamsters either injected with copper or fed a diet high in copper (Aulerich et al., 1982, as cited in ATSDR, 1990). Developmental effects have been observed in minks administered doses 50 times lower than those given to mice and hamsters. Furthermore, distinct developmental effects in minks have resulted from dietary levels of copper that were well below the levels that would have been expected to cause maternal toxicity. Although developmental effects have not been reported in humans, there is a possibility that there may be an increased incidence of spontaneous abortion and miscarriage in women exposed to high levels of copper (ATSDR, 1990).

Reproductive Toxicity

Although reproductive performance was not adversely affected in minks fed a diet high in copper (Aulerich et al., 1982, as cited in ATSDR, 1990), the insertion of copper wires into the vas deferens or uterus prior to conception or at gestational day 3 resulted in decreased fertility or decreased number of implantation sites in monkeys, rats, hamsters, and rabbits (Chang and Tatum, 1970, as cited in ATSDR, 1990).

Genotoxicity

Several in vitro studies have examined genotoxic effects of copper in nonhuman systems. The results of tests using prokaryotic organisms are equivocal. However, positive results have been observed in vitro and in vivo in mammalian systems. At low levels (0.01 - 0.1 mM Cu as copper sulfate), DNA strand breaks were not observed in rat hepatocytes; however strand breaks did occur at high concentrations (0.04 mM Cu as copper sulfate) (Sina et al., 1983, as cited in

ATSDR, 1990). Although there are no data on the mutagenicity of copper in humans, in vivo studies and mammalian system in vitro studies suggest that copper is a potential human mutagen.

Carcinogenicity

An elevated incidence of cancer has not been observed in humans or animals exposed to copper via inhalation, oral, or dermal routes of exposure. The US EPA classifies copper as a Class D carcinogen (not classified) based on the fact that there are no human data, inadequate animal data from assays of copper compounds, and equivocal mutagenicity data (IRIS, 1995).

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1,3-DICHLOROBENZENE

(CAS RN 541-73-1)

1,3-Dichlorobenzene (synonym=meta- [or m-]dichlorobenzene) is a colorless liquid which is not known to occur in nature (HSDB, 1995). It is soluble in alcohol, ether, acetone and benzene (Weast, 1988, as cited in HSDB, 1995). 1,3-Dichlorobenzene is used as a fumigant and insecticide and in the preparation of arylene sulfide polymers in a polymerization process (Sax, 1987, and Kirk-Othmer Encyclopedia of Chemical Technology, 1984, both as cited in HSDB, 1995). 1,3-Dichlorobenzene has a boiling point of 173.53°C, and a melting point of -24.7°C (Weast, 1988, as cited in HSDB, 1995).

Absorption/Distribution/Metabolism/Excretion

The dichlorobenzenes may be absorbed through the lung, GI tract, and intact skin. Relatively low water solubility and high lipid solubility favor their penetration of most membranes by diffusion, including pulmonary and GI epithelia, the brain, hepatic parenchyma, renal tubules, and the placenta (USEPA Ambient Water Quality Criteria Document, 1980, as cited in HSDB, 1995). When fed to rabbits, m-dichlorobenzene yielded glucuronides (31%), sulfates (11%), mercapturic acid (9%), and catechols (4%); 2,4-dichlorophenylmercapturic acid and 3,5-dichlorocatechol were also observed. (Menzie, 1978, as cited in HSDB, 1995). 1,3-Dichlorobenzene was reported to be among several metabolites of gamma-pentachloro-1-cyclohexane in corn and pea seedlings (USEPA Ambient Water Quality Criteria Document, 1980, as cited in HSDB, 1995).

Dichlorobenzene (all isomers) was identified in 100% of 42 samples of human breast milk collected in five urban areas of the United States at concentrations of 0.04 - 68 ppb (Erickson et al., 1980, as cited in ATSDR, 1992)

General population exposure to 1,3-dichlorobenzene may occur through oral consumption of contaminated drinking water and food (particularly fish), especially in the vicinity of effluent discharges such as Lake Ontario and the Great Lakes. General population exposure may also occur through inhalation of contaminated ambient air, since 1,3-dichlorobenzene has been detected in many areas of the United States. Occupational exposure probably occurs during the manufacture of dichlorobenzenes and the uses of 1,3-dichlorobenzene as a fumigant, chemical intermediate, and solvent. Probable routes of occupational exposure are inhalation of contaminated air and dermal contact (HSDB, 1995).

Acute Toxicity

The acute toxicities of chlorobenzenes to rainbow trout (*Salmo gairdneri*) as intraperitoneal injection lethality, serum sorbitol dehydrogenase activity, and 96-hour lethal concentration were investigated in terms of quantitative structure-activity correlations. The effects were primarily related to the octanol/water partition coefficients (log P) (Kaiser et al., 1984, as cited in HSDB, 1995).

In two subjects with chronic lymphoid leukemia, one had been exposed to glue containing 2% ortho-dichlorobenzene from 1945-1961 and the other had been exposed from 1940 to 1950 to solvent containing ortho-dichlorobenzene (80%); the actual carcinogenic agent in these exposures has not been identified (Girard et al., 1969, as cited in HSDB, 1995).

Target Organ Toxicity

Vapors and sprays containing dichlorobenzenes are irritating to eyes, nose, and throat, but the effect seems to disappear quickly. When swallowed they cause burning pain in the stomach, nausea, vomiting, and diarrhea. Hemoglobin may change to methemoglobin with resulting dusty color of skin. The liver and kidney may be damaged (Thienes et al., 1972, as cited in HSDB, 1995).

The US EPA has commented that the chronic oral RfD is considered not verifiable by the RfD/RfC Work Group (HEAST, 1997). The EPA IRIS database also lists no available chronic oral RfD (IRIS, 2000).

Carcinogenicity

The US EPA classifies 1,3-dichlorobenzene as a Class D carcinogen (not classified) based on the fact that there are no human data, no animal data, and limited genetic data (IRIS, 1995).

REFERENCES

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DIOXIN/FURAN

No data were located regarding the pharmacokinetics of the polychlorinated dibenzo-p-dioxins (PCDDs) or (PCDFs) of concern; however, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) has been used as a surrogate for other structurally similar members of these chemical classes.

Absorption/Distribution/Metabolism/Excretion

Estimates of the GI absorption of TCDD ranged from 50 to 86 percent of the administered dose in rats; comparable data were obtained for rats and hamsters. In rats treated dermally with 26 ng TCDD in methanol dermal absorption after 24 hours approximated 40 percent of that absorbed by the GI tract after an equivalent dose in ethanol. Dermal absorption from vaseline or polyethylene glycol vehicles was substantially less than from methanol, but quantitative estimates cannot be made from the available data.

Target Organ Toxicity

In rodents given single oral or intraperitoneal doses, or treated for two years with TCDD in the diet, the highest concentrations and greatest tissue depots occurred in the liver, followed closely by adipose tissue. Concentrations in other tissues were considerably lower than those in fat. Mouse liver continued to sequester TCDD more efficiently with prolonged exposure. In nonhuman primates and guinea pigs, however, greater TCDD concentrations and tissue depots occurred in the adipose tissue than in the liver. Data obtained at necropsy from one woman potentially exposed to TCDD, showed concentrations in adipose tissue about an order of magnitude higher than levels in the liver. Radioactivity from intravenous dosing with [^{14}C]2,3,7,8-TCDD has been shown to cross the placenta of rats and mice; concentrations of fetal tissues were lower than in maternal tissues

In rodents and guinea pigs, TCDD was metabolized by microsomal mixed-function oxidase enzymes to hydroxylated derivatives that were conjugated with glucuronide or sulfate for excretion via the bile or urine, respectively. The hydroxylation of several different PCDDs in the rat was postulated to involve formation of arene oxide intermediates (EPA, 1985). In rats, the metabolism of TCDD was inducible but relatively slow, about four orders of magnitude slower than the metabolism of benzo(a)pyrene. There was considerable species variation in the rate of metabolism of TCDD.

Studies with [^{14}C]2,3,7,8-TCDD showed that fecal excretion accounted for 39 to 99 percent of the total (fecal and urinary) excretion of radioactivity (EPA, 1985). Elimination half-lives (assuming first order kinetics) ranged from 11 to 30 days, inversely correlated with species sensitivity to TCDD. There was considerable interspecies variation in the relative importance of fecal versus urinary excretion and in the elimination half-lives.

The only effect in humans clearly attributable to TCDD was chloracne (ATSDR, 1989). The available data, however, also associated exposure to TCDD with hepatotoxicity and neurotoxicity in humans. In animals, TCDD toxicity is most commonly manifested as a wasting syndrome with thymic atrophy terminating in death, with a large number of organ systems showing nonspecific effects. Chronic treatment of animals with TCDD or a mixture of two isomers of hexachlorodibenzo-p-dioxin resulted in liver damage. Immunologic effects may be among the more sensitive endpoints of exposure to the PCDDs in animals TCDD is a developmental and reproductive toxicant in animal models.

Carcinogenicity

The EPA (1992) has verified dibenzofuran as a cancer weight-of-evidence Group D compound (not classifiable as to carcinogenicity to humans), based on a lack of cancer data in humans or animals. Data regarding human carcinogenicity of TCDD obtained from epidemiologic studies of workers exposed to pesticides or to other chlorinated chemicals known to be contaminated with TCDD, are conflicting (ATSDR, 1989). The interpretation of these studies is clouded,

because exposure to TCDD was not quantified, multiple routes of exposure (dermal, inhalation, oral) were involved, and the workers were exposed to other potentially carcinogenic compounds. TCDD, however, is clearly carcinogenic in animal models, inducing thyroid, lung, and live tumors in orally treated rats and mice (EPA, 1985) Similarly, oral treatment with a mixture of two hexachlorodibenzo-p-dioxin isomers induced liver tumors in rats and mice. On the basis of the animal data, TCDD and the hexachlorodibenzo-p-dioxins were assigned to EPA cancer weight-of-evidence Group B2 (probably human carcinogen). Although the PCDDs and PCDFs of concern were not classified, they are treated as probable human carcinogens, for which slope factors are derived.

The EPA (1993) has presented provisional oral and inhalation slope factors for TCDD of 150,000†mg/kg/day, based on the incidence of liver and lung tumors in an oral study in rats. HEAST (1997) also lists the 150,000 mg/kg/day oral and inhalation slope factors. In the absence of satisfactory congener-specific cancer data, the EPA (1989) derived toxicity equivalency factors (TEFs) for the other PCDDs and PCDFs, by assuming that all manifestations of toxicity for all members of these classes are mediated by a common mechanism (i.e., binding to the intraculluar AH receptor of target cells).

Considerable uncertainty surrounds the carcinogenic potential of the PCDDs and PCDFs of concern. Although TCDD is classified as a weight-of-evidence Group B2 substance, the homologues of concern are not classified. The appropriateness of estimating cancer potency of (i.e., regulating as carcinogens) compounds not assigned to a cancer weight-of-evidence group is questionable (EPA, 1986).

There is uncertainty about the slope factor for TCDD. Additional uncertainty is introduced by the use of the TEFs themselves, most of which were derived not from cancer data, but from in vitro data such as enzyme induction, which is only hypothetically related to a carcinogenic role. For example, the TEF of 0.001 for octachlorodibenzo-p-dioxins (OCDDs) and octachlorodibenzofurans (OCDFs) is based on the appearance of "dioxin-like" effects and detectable levels of OCDD late in a 13-week study of male rats treated with OCDD (Couture et

al., 1988) and on in vitro evidence of enzyme induction (EPA, 1989). Before the Couture et al. (1988) paper was available, the TEF for these homologues, based on limited in vivo and in vitro data was 0.0.

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LEAD

(CAS RN 7439-92-1)

Lead is a naturally occurring bluish-grey metal found in small amounts in the earth's crust. It is found in plants and animals, air, drinking water, rivers, lakes, oceans, dust, and soil. Lead remains in soil for many years. Mixed ores or recycled scrap are the source of lead to industry. Its main use is in the manufacture of storage batteries followed by production of chemicals including paints, gasoline additives, various metal products, and ammunition.

The largest source of lead in air is from vehicle exhaust. Other sources include emissions from iron and steel production, smelting operations, municipal waste incinerators, and lead-acid-battery manufacturers. Lead is also present in tobacco smoke. The main sources of lead released to water are lead plumbing and solder in buildings; lead-containing dust and soil carried into water by rain and wind; and wastewater from industries using lead.

Exposure to lead may occur via breathing air, drinking water, and eating soil or foods that contain lead. Another source, for children, is from swallowing non-food items such as chips of lead-containing paint (i.e., pica).

Absorption/Distribution/Metabolism/Excretion

The rate and degree of lead absorption are largely related to its solubility in body tissues and fluids, among other factors. Upon inhalation, some fraction of inhaled airborne and orally ingested lead is deposited in the respiratory tract. The rate of deposition in adult humans is approximately 30 percent to 50 percent. Upon deposition in the lower respiratory tract, all chemical forms of lead are almost completely absorbed. Approximately 20 percent of inhaled lead was absorbed within 1 hour, and 70 percent was absorbed within 10 hours in humans breathing lead-containing engine exhausts or lead oxide and lead nitrate aerosols at 2 to 10†µg/m^3 lead (Chamberlain et al., 1978 cited in ATSDR, 1986). Inhaled lead is absorbed extensively and rapidly in animals as well. Morgan and Holmes (1978 cited in ATSDR, 1986)

estimated absorption rates of 50 percent within 1 hour and 98 percent within 7 days in adult rats breathing the equivalent of 6 mg/m^3 lead.

The primary site of lead absorption in children is the GI tract (approximately 50 percent) upon oral exposure, compared to 8 percent in adults (Hammond, 1982 cited in ATSDR, 1986) or 15 percent (Chamberlain et al., 1978 cited in ATSDR, 1986). Absorption may be as high as 45†percent in fasting adults (Chamberlain et al., 1978 cited in ATSDR, 1986). GI absorption in children (from nonfood sources) is estimated to be approximately 30 percent from dirt/dust and 17 percent from paint chips (Drill et al., 1979 cited in ATSDR, 1986). Absorption values are similar in animals (i.e., 1 percent to 15 percent in adults) The extent of GI absorption is age dependent (i.e., younger animals absorb 40 to 50 times more lead via the diet than do adults). GI absorption is enhanced by milk products, low calcium and vitamin D levels, fasting, or iron deficiencies. Dermal absorption is much less significant due to a greatly reduced dermal absorption rate. In humans, approximately 0 percent to 0.3 percent absorption has been measured (ATSDR, 1986).

Once in the body, lead is dispersed primarily to the bone, blood, and soft tissue pools regardless of the route of absorption. The half-life of lead in blood is approximately 36 days, in soft-tissue is 40 days and in bone is 10^4 days. Greater than 99 percent of blood lead is associated with the red blood cells. Over 50 percent of this blood pool is bound to hemoglobin, with lesser amounts bound to other proteins. Fetal hemoglobin has a greater affinity for lead than does adult hemoglobin. The biological half-life of lead in the blood of 2 year-old children is approximately 10 months.

In human adults approximately 95 percent of the total lead body burden is present in the bones. In children, this value is 73 percent. Bone lead levels increase with age. In most soft tissue, lead does not appear to accumulate as a function of age in humans over 20 years old.

Any dietary lead not absorbed by the GI tract is eliminated in the feces of both humans and animals. Rosen (1985 cited in ATSDR, 1986) demonstrated that 50 percent to 60 percent of the

absorbed fraction of lead was excreted on a short-term basis. Chamberlain et al. (1978 cited in ATSDR, 1986) found this half-life to be 19 days. Infants have been shown to retain 34 percent of the total amount of absorbed lead whereas adults demonstrate a 1 percent retention rate. In animals (rats) initial excretion occurs in the urine followed by greater excretion in the feces. In rats excretion occurs in two phases. The initial phase half-life is 21 hours followed by a slower 280 hour second phase (Morgan et al., 1977 cited in ATSDR, 1986).

Acute Toxicity

Human Toxicity. Mortality for workers was studied by Cooper et al. (1986 cited in ATSDR, 1986) in a cohort study of employees at lead-producing facilities. Two cohorts, all of whom had been employed for at least 1 year during 1946 through 1970 were studied for mortality from 1947 through 1980. Mean blood lead levels were 63 µg/dL for 1,326 of the 4,519 battery workers and 80 µg/dL for 537 of 2,300 lead production workers from 1947 through 1972. The number of observed deaths of both groups was significantly greater (P less than 0.01) than expected. Increased mortality resulted in a large part from malignant neoplasms; chronic renal disease; and "ill-defined" causes.

In children, lead intoxication leading to encephalopathy and death may result from oral exposure. The range of blood lead levels associated with encephalopathy is approximately 90 to 700 or 800 µg/dL and the range associated with death is approximately 125 to 750 µg/dL. The mortality rate of untreated lead encephalopathy (a severe neurological effect) in children was estimated by the EPA to be 65 percent prior to the introduction of chelation therapy. Shortly after therapy is started, mortality decreases for 25 to 33 percent. An absence of signs or symptoms has been observed in some children at blood lead levels of 60 to 300 µg/dL. Acute lead poisoning symptoms other than signs of encephalopathy have been observed at blood lead levels of approximately 60 to 450 µg/dL. Rummo (1974 cited in ATSDR, 1986) and Rummo et al. (1979 cited in ATSDR, 1986) observed hyperactivity and a decrease of 16 IQ points on the McCarthy General Cognitive Index (GCI) among children who previously had encephalopathy and whose mean blood lead levels at the time of encephalopathy were 88†µg/dL.

Lead interferes with heme biosynthesis by altering the activity of several key enzymes A marked interference results in a reduction of the hemoglobin concentration in blood. Decreased hemoglobin production, coupled with an increase in erythrocyte destruction, results in anemia.

In adults, early symptoms of lead encephalopathy may develop within weeks of initial exposure. These symptoms include dullness, irritability, poor attention span, headache, muscular tremor, loss of memory, and hallucinations. The condition may then worsen to delirium, convulsions, paralysis, coma, and death. Severe lead encephalopathy is not observed except at blood lead levels well in excess of 120 µg/dL (Kehoe, 1961a, b, c cited in ATSDR, 1986). Smith et al. (1938 cited in ATSDR, 1986) suggested that acute lead poisoning in adults can occur at blood lead levels of approximately 100 µg/dL. Increased central and peripheral nervous system and GI symptoms were noted in 25 lead workers with maximum blood lead levels of 50 to 69 µg/dL (Haenninen et al., 1979 cited in ATSDR, 1986). Decreases in nerve conduction velocity have been observed in workers with blood lead levels of 30 to 48 µg/dL (Seppalainen et al., 1983 cited in ATSDR, 1986). Robinson et al. (1985 cited in ATSDR, 1986) cited evidence of a lead-related decrease in hearing acurity of children at maximum blood lead levels of 6.2 to 56.0 µg/dL. Lead neuropathy in children may be associated with the incidence of sickle cell disease in children

Evidence from occupational, clinical, and general population studies indicates that lead affects the cardiovascular system in humans, resulting in cardiac lesions, electrocardiographic abnormalities, and increases in blood pressure. Renal toxicity (early or acute nephropathy) has also been associated with lead exposure but appears to be reversible. Chronic lead nephropathy associated with blood lead levels of 40 to greater than 100 µg/dL is irreversible.

Animal Toxicity. High rates of mortality were observed in rats exposed to tetramethyl lead (organic lead) for 7 h/day, 5 days/week at 63 mg/m^3 for 10 days, 49 mg/m^3 for 18 days, and 22 mg/m^3 for 35 days (Davis et al., 1963 cited in ATSDR, 1986). Several studies in animals also indicate that the effects of lead on heme synthesis occur in many tissues. The formation of heme-containing cytochromes is inhibited in animals treated intraperitoneally or orally with lead

compounds. Impairment of heme synthesis results in disruption of a variety of important physiological processes including reduced hemoglobin synthesis, reduction of hemoproteins, renal endocrine effects, and hepatic effects

Studies in animals have shown delays in reflex development in rats during early postnatal life at greater than 59 µg/dL (Kishi et al., 1983 cited in ATSDR, 1986). Decreased visual acuity in young rats have been observed in several studies at mean blood lead levels of 65 µg/dL. Blood lead levels as low as 15 to 20 µg/dL were associated with slower learning and higher rates of inappropriate responses (Cory - Slechta et al., 1985; Schlipkoter and Winneke, 1980 cited in ATSDR, 1986).

Exposure of male rats had an effect on cardiovascular function at levels of 50 ppm lead. Lead acetate was given in drinking water for 160 days with results of markedly increased blood pressure (182/138) as compared to 129/98 in controls (Iannaccone et al., 1981 cited in ATSDR, 1986). The mean blood lead level in the treated group was 38.4 µg/dL.

Animal studies provide evidence of nephropathy similar to that in humans, particularly after acute exposures. Lead appears to affect vitamin D metabolism in renal tubule cells such that circulating levels of the vitamin D hormone, 1,25 - dihydroxyvitamin D, are reduced (Smith et al., 1981 cited in ATSDR, 1986). High calcium diets protected against this effect. In vitro studies with rat pituitary cells showed that lead inhibited the thyrotropin-releasing hormone (TRH) and stimulated release of thyrotropin-stimulating hormone (TSH) in a dose-related manner (Huseman et al., 1987 cited in ATSDR, 1986). This supports the conclusions drawn from human data.

Developmental and Reproductive Toxicity

The EPA has concluded, based on a review of several studies, that a definitive association between prenatal lead exposure in humans and the occurrence of congenital anomalies has not been demonstrated. Various studies have reported decreased birth weights, still births, and

placental weight. However, as noted by EPA, the majority of these studies did not account for possible confounding variables (i.e., socioeconomic status and concurrent exposures). Therefore, the above conclusion has been made. It has been noted, however, that gestational age may be reduced as prenatal lead exposure increases, even at blood lead levels below 15 µg/dL. Several studies have indicated negative correlations between maternal or cord blood lead levels and gestational age.

In one prospective study of the effects of pre- and postnatal lead exposure on child development McMichael et al. (1986 cited in ATSDR, 1986) studied 831 pregnant women and followed 774 pregnancies to completion in a lead smelter town. Blood lead levels were significantly higher in women who lived in the town compared to those residing outside the town (11.2 µg/dL versus 7.5 µg/dL). No association between lead exposure and congenital anomalies was noted when factors such as smoking and alcohol consumption were reviewed. Also observed was a greater incidence of low-birth weight (less than 2,500 g at gestational age greater than or equal to 37 weeks) in the town. More miscarriages and stillbirths occurred in the Port Pirie mothers compared to others. Neurobehavioral development, therefore, appears to be deleteriously affected by prenatal lead exposures. Transplacental transfer of lead in humans has been demonstrated in a number of studies. Fetal uptake of lead occurs by the 12th week of development and increases throughout development (Barltrop, 1969; Horiuchi et al., 1959 cited in ATSDR, 1986). Highest lead levels were measured in fetal bone, kidney, and liver tissue and lesser amounts in the brain and heart.

Teratogenicity studies in rats and mice provide no evidence that lead compounds (acetate or nitrate) are teratogenic when exposure occurs by natural routes. Intravenous or intraperitoneal injection of lead compounds into pregnant rats, mice or hamsters, however, has produced malformations in several studies. There are indications, as well, that lead accumulates in the placenta in times of fetal stress.

Severe occupational exposure (primarily via inhalation) to lead has been shown to be associated with a high likelihood of spontaneous abortions. The EPA has concluded that reproduction

effects on the sperm or testes of men chronically exposed at blood lead levels of 40 to 50 µg/dL may occur. Lancranjan et al. (1975 cited in ATSDR, 1986) studied 150 men with long-term lead exposure, categorized as follows: lead-poisoned (mean blood lead level 74.5 µg/dL); moderately poisoned (mean: 52.8 µg/dL); slightly poisoned (mean: 41 µg/dL); or physiologically poisoned (mean: 23 µg/dL). The lead-poisoned and moderately exposed groups had decreases in fertility. The effects were thought to be directly on the testes.

Chowdhury et al. (1984) found testicular atrophy and cellular degeneration in male rats given lead acetate in drinking water at 1 g/L (1,000 ppm) for 60 days. Blood lead levels averaged 142.6 µg/dL.

Genotoxicity

Results of in vitro studies with human lymphocyte cultures performed with lead acetate have been nearly divided between positive and negative. Results of in vivo studies are also contradictory, although the weight of evidence does suggest that lead has an effect on chromosomes. The status of calcium nutrition may be important in the expression of lead-induced clastogenicity in both in vitro and in vivo tests. Studies in mice (DeKnudt and Gerber, 1979 cited in ATSDR, 1986) and monkeys (DeKnudt et al., 1977 cited in ATSDR, 1986) indicated that calcium deficiency may enhance the genotoxicity of lead.

Carcinogenicity

The EPA has concluded that current human data are inadequate to refute or demonstrate the potential carcinogenicity of lead exposure to humans. Animal data, however, were deemed sufficient (particularly soluble lead salts).

An extensive series of reports of a large number of workers at six domestic lead production plants (smelter and recycling plants) and ten battery plants exists. Increased incidences of total malignant neoplasms were observed for both categories of lead workers. In a more recent

evaluation of a more select subset from the original study increases in total malignancies in both groups of workers attributed to digestive and respiratory cancers was reported (Cooper and Gaffey, 1975; Cooper, 1976 cited in ATSDR, 1986).

The most comprehensive set of studies of carcinogenicity resulting from oral exposure to lead was performed by Azar et al. (1973 cited in ATSDR, 1986). Lead acetate was given to rats at 0, 10, 50, 100, 500, 1,000, or 2,000 ppm lead in the diet for 2 years. Fifty rats per sex were exposed. After the study was in progress, groups of 20 rats per sex were also fed 0, 1,000 or 2,000 ppm lead in diet. Blood lead levels measured at 24 months were 12.7, 11.0, 18.5, 35.2 and 77.8 µg/dL in the 0-, 10-, 50-, 100-, and 500- ppm groups. Levels of 16.4, 98.6, and 98.4†µg/dL were observed in the 0-, 1,000-, and 2,000- ppm groups. Renal tumors occurred in 5/50 male rats at 500 ppm, in 10/20 males at 1,000 ppm, and in 16/20 males and 7/20 females at 2,000 ppm.

The EPA has classified lead in Class B2 - Probable Human Carcinogen. However, the agency is currently reviewing both the toxicity and carcinogenicity of lead for further evidence. Cancer slope factors and unit risk factors are not available.

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MANGANESE

(CAS RN 7439-96-5)

Manganese is a naturally occurring substance found in many types of rock. Pure manganese is a silver-colored metal that is not naturally occurring, but occurs combined with other chemicals such as oxygen, sulfur, and chlorine. Some manganese compounds can dissolve in water, and low levels of these compounds are naturally present in lakes, streams, and the ocean. Rocks containing high levels of manganese compounds are mined for use in the production of manganese metal. This metal is mixed with iron to make steel.

Absorption/Distribution/Metabolism/Excretion

A significant fraction of manganese particles inhaled are initially deposited in the lungs and then transported via mucociliary transport to the GI tract. Thus, manganese may be absorbed both in the lungs and the GI tract. The amount absorbed across the human GI tract is typically 3 to 5 percent (Davidsson et al., 1988, 1989; Mena et al., 1969 cited in ATSDR, 1991b). Low iron levels apparently lead to increased manganese absorption. GIuptake in animals following oral exposure is similar, with uptake estimated at 2.5 to 5.5 percent in rats (Pollack et al., 1965 cited in ATSDR, 1991b). Several studies in animals indicate that GI absorption may vary with age. Rehnberg et al. (1980, 1981 cited in ATSDR, 1991b) noted that rats exposed to manganese tetroxide showed larger increases in tissue levels in neonatal rats (ages 1 to 15 days) than in older rats.

In humans, most tissue concentrations of manganese range from 0.1 and 1 μg per gram wet weight, with highest levels observed in the liver, pancreas, and kidneys, and lowest levels in bone and fat (Sumino et al., 1975; Tipton and Cook, 1963 cited in ATSDR, 1991b). Levels in animal tissue are similar. Studies in animals indicate that upon oral exposure, an increase in manganese levels was observed in all tissues; however, the magnitude of the increase diminishes over time (Kristensson et al., 1986; Rehnberg et al., 1980, 1981, 1982 cited in ATSDR, 1991b).

Excretion of manganese in humans and animals is primarily through the feces. Humans who inhaled manganese as manganese tetroxide or manganese chloride excreted approximately 60 percent of the material deposited in the lungs in the feces within 4 days (Mena et al., 1969 cited in ATSDR, 1991b).

Acute Toxicity

Human Toxicity. Inhalation of particulate manganese compounds leads to an inflammatory response in the lungs. Damage to lung tissue is usually not extensive, but may include local areas of edema, fibrosis, or emphysema (Suzuki et al., 1978; Davies, 1946; Shiotsuka, 1984; Zaidi et al., 1973 cited in ATSDR, 1991b). Symptoms and signs of lung irritation and injury may include cough, bronchitis, and minor reductions of lung function. The EPA notes that an inflammatory response of this type is not unique to manganese-containing particles, but it is characteristic of nearly all inhalable particulate matter. An increased prevalence of infectious lung disease, pneumonia in particular, has been noted in workers with chronic occupational exposure to manganese dust and in residents near a ferromanganese factory (Davies, 1946; WHO, 1987 cited in ATSDR, 1991b).

The EPA has published an oral RfD of 1 4E-01 mg/kg/day for manganese in food (IRIS, 2000). The UF is 1, and the RfD is based on CNS effects from chronic human ingestion. WHO (1973, as citedin ATSDR, 1991b) reported no adverse effects in humans upon consumption of 8 to 9 mg Mn/day associated with daily diets. Evaluation of standard diets from the United States, England, and Holland revealed average daily intakes of 2.3 to 8.8 mg Mn/day (Schroeder et al., 1966, as cited in ATSDR, 1991b). This study suggested that higher levels may be achieved from a normal diet (especially a vegetarian diet). Schroeder et al. reported a chronic human NOAEL of 11.5 mg Mn/day as safe. And finally, the National Research Council (NRC, 1989, as cited in ATSDR, 1991b) determined an "adequate and safe" intake of manganese to be 2 to 5 mg/day for adults.

The EPA has published an inhalation RfC of 5.0E-05 mg/m^3 (IRIS, 2000) which yields an inhalation RfD of 1.4E-05 mg/kg-day for manganese. The RfC is based on impairment of neurobehavioral function and has a UF of 1000. This UF results from a factor of 10 for sensitive individuals; 10 for use of a LOAEL; and 10 for the less-than-chronic periods of exposure, lack of developmental data, and potential unquantified differences in toxicity of various forms of manganese.

Animal Toxicity. Inhalation of particulate manganese compounds also leads to an inflammatory response in animal lungs. Increased susceptibility to lung infection by bacterial pathogens following inhalation of manganese dusts has been confirmed in several acute animal studies (Adkins et al., 1980; Maigetter et al., 1976 cited in ATSDR, 1991b).

Most acute toxicity studies indicate that manganese compounds have low oral acute toxicity. Upon daily oral exposure of 930 mg/kg-day, significant mortality in rats was not observed until after 16 months of exposure (Hejtmancik et al., 1987a cited in ATSDR, 1991b). Chronic exposure of mice to 810 mg/kg-day did not cause increased mortality within 24 months (Hejtmancik et al., 1987b cited in ATSDR, 1991b). Gianutsos and Murray (1982 cited in ATSDR, 1991b) administered oral doses of manganese (as manganese chloride) to laboratory animals as high as 2,300 mg/kg-day for six months without observing lethality.

Developmental and Reproductive Toxicity

The effects of manganese on fetal development in humans have not been investigated thoroughly. The incidence of stillbirths and malformations has been studied in an Australian population living on an island where high environmental levels of manganese existed (Kilburn, 1987 cited in ATSDR, 1991b). However, because the study population was small and data from a suitable control group was lacking, it is not possible to judge if the incidence of abnormalities was higher than average.

The number of children born to occupationally exposed males may be lower than average (Lauwreys et al., 1985 cited in ATSDR, 1991b), and decreased libido and impotence are frequently observed in male workers exposed to high levels of manganese dusts in the work place (Emara et al., 1971; Mena et al., 1967; Rodier, 1955 cited in ATSDR, 1991b). Young male rats and mice exposed orally to manganese experienced delayed growth and maturation of the testes and other reproductive tissues (Gray and Laskey, 1980 cited in ATSDR, 1991b). Information on reproductive effects in females is very limited. One study indicated, however, that female rats exposed to manganese oxide by inhalation for 18 weeks had an increased number of pups per litter (ATSDR, 1991b).

Genotoxicity

Results of manganese genotoxicity in vitro have been mixed, while those from in vivo tests have been negative. Errors in the fidelity of DNA replication have been noted in vitro as Mn(+2) can substitute for the magnesium ion (Mg+2) in DNA polymerase (El-Deiry et al., 1984 cited in ATSDR, 1991b).

Carcinogenicity

Information on the carcinogenic potential of manganese is limited. The EPA has designated manganese as a class D component (IRIS, 6/95). In rats, males exposed to 86, 290, or 930 mg/kg-day manganese (as manganese sulfate) had an increased incidence of pancreatic cell adenomas and carcinomas (Hejtmancik et al., 1987a cited in ATSDR, 1991b). However, it should be noted that the incidence was low and was not dose-responsive. It should be noted further that this tumor type only appeared in one female (in the mid-dose group) and that no increases in tumor frequency were detected in other tissues.

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MERCURY

(CAS RN 7439-97-6)

Mercury is a transition metal and, in its elemental state, occurs at room temperature as a liquid. It is commonly produced as a byproduct of gold mining and is produced from mining operations. It is commonly used in thermometers, barometers and other pressure-sensing devices and in electrical components. World production of mercury exceeds 6.5 million kilograms annually.

Adsorption/Distribution/Metabolism/Excretion

Mercury occurs naturally in the environment and is distributed throughout the world. It occurs in three valence states; as a metal and various inorganic and organic complexes. It is discharged into the environment in the form of gaseous emissions, various salts in industrial processes, and is capable of bioaccumulation into terrestrial and aquatic food chains. A saturated atmosphere of elemental mercury can contain up to 18 mg/m^3 and it can exist in the monoatomic state as a vapor. Inorganic mercury (Hg^{+1} and Hg^{+2}) readily forms complexes with organics, especially sulfhydryl groups and can form salts with organic and inorganic acids. Organic mercury consists of primarily methyl, ethyl, phenyl and alkoxyalkyl mercury compounds in the form of organic and inorganic acid salts. The compounds readily bioaccumulate due to their lipophilic nature and can interact with biologically significant ligands. These compounds are capable of passing placental barriers.

Acute Toxicity

Acute human exposure to high concentrations of mercury vapor results in loss of respiratory function and death due to pulmonary tissue necrosis and edema (Teng and Brennan, 1959). Organic mercury exposure involving diethylmercury at concentrations up to 1 mg/m^3 for up to four months resulted in death with major tissue damage being in the gastrointestinal tract. Acute inhalation exposure of mercury vapor and organic mercury to experimental animals results in death due to pulmonary edema.

Inhalation of metallic mercury vapor results in systemic toxicity to humans and experimental animals. The effects are dose related such that at low levels, the central nervous system and kidney are target organs while at higher doses, the respiratory tract, cardiovascular and gastrointestinal organs are targeted. Respiratory effects in humans and rodents include pulmonary edema, lobar pneumonia, desquamation of bronchiolar epithelium, necrosis and death (Ashe et al., 1953). Cardiovascular effects include initial increase in blood pressure followed by degeneration and myocardial necrosis. Gastrointestinal effects include nausea, vomiting, gingivitis, mercurial stomatitis and necrosis of the intestinal mucosa (Lillis, 1985). Renal effects include proteinuria, hematuria, degeneration of the convoluted tubules and death (Campbell, 1948). Creatine excretion increased in exposed individuals with increasing dose suggesting that this marker might be a useful gauge for the level of mercury exposure (Buch et al., 1980). In rodents, renal tubular epithelium degeneration is noted at moderate doses (3 mg/m^3) (Kishi et al., 1978). Based on these studies, a level of from 1 to 3 mg/m^3 resulted in a lowest-observable-adverse-effects-level (LOAEL).

Target Organ Toxicity

The central nervous system is a major target organ for elemental mercury exposure. Elemental mercury rapidly passes the blood-brain barrier and can exert toxicity based on conversion to inorganic forms and interaction with sulfhydryl groups in the neuronal tissue. Acute exposure results in excitability, tremors, decreased motor and muscular function, headache, visual disturbances, ataxia, dysarthria and, with severe exposure, paralysis and death (Cassarett and Doull, 1986; Hanninen, 1982). Chronic exposure to elemental mercury results in tremors, loss of short-term memory, decreased psychomotor skill and general neurological dysfunction that becomes irreversible after prolonged exposure (Fawer et al., 1983). Elevated urinary excretion of mercury generally correlates with neurological symptoms. Due to the cumulative nature of mercury intoxication, a chronic inhalation MRL of 0.00026 mg/m^3 has been suggested for a long-term exposure level of atmospheric mercury.

Developmental Toxicity

Rodent exposure to metallic mercury results in neurological and behavioral effects similar to those manifested in humans, although rodents appear less sensitive than humans to mercury inhalation (Armstrong, 1963; Ganser and Kirschner, 1985).

Investigations in humans following prenatal exposure to mercury suggests increased spontaneous abortion and menstrual disturbances. Due to the rapid absorption of mercury via inhalation, transfer of mercury from maternal blood to maternal milk can result in neonatal exposure to mercury (Cassarett and Doull, 1986).

Elemental mercury vapor caused increased fetal toxicity in rodents. The number of resorptions and fetal mortality increased with treatment groups. Rats exposed to doses of 0.5 mg/m^3 during days 10 to 15 of gestation resulted in increased resorptions and increased skeletal malformations (Steffek et al., 1987). Based on rodent and human data, mercury exposure during mid to late pregnancy could result in developmental and/or reproductive toxicity.

Genotoxicity

Genotoxic effects of elemental and inorganic mercury via inhalation include elevated chromosomal abnormalities in lymphocytes. Conflicting reports indicate the potential for development of both chromosomal aberrations and aneuploidy in humans. Bone marrow chromosomal aberrations have been demonstrated in mice exposed to organic mercury vapors. Short-term mutagenicity assays in various cultured cells indicate a lack if direct mutagenic activity of mercury or mercury salts.

Carcinogenicity

Based on fairly extensive human epidemiological investigations, there is no correlation between occupational mercury exposure and increased tumor burden. There is no experimental animal data or bioassays that indicated that the various forms of mercury could enhance tumor development in rodents. Based on these data, the Environmental Protection Agency (EPA) has classified mercury as a class D compound and has not developed cancer potency values for mercury (IRIS, 1994).

Chronic Toxicity

Chronic exposure to elemental mercury vapor results in prolonged neurological symptoms which include tremors, loss of memory, decreased psychomotor skill and neurological dysfunction (Fawer et al., 1983). Increased urinary excretion of mercury and the development of proteinuria indicate chronic mercury intoxication as does the elevated creatine excretion (Ganser and

Kirschner, 1985). The EPA (IRIS) database lists a chronic inhalation reference dose (RfC) of 3.0×10^{-4} mg/m^3 for elemental mercury. A UF value of 1000 was applied. The EPA (IRIS) database also lists a chronic oral reference dose (RfD) for mercuric chloride ($HgCl_2$) of 3.0×10^{-4} mg/kg-day (IRIS, 2000), which is also used as the oral reference dose for 'mercury and compounds' in the EPA Region IX PRG tables (EPA, 1999).

Toxicokinetics

Absorption is high for inhalation of elemental mercury vapor, but low for ingestion of elemental mercury. Oral absorption of inorganic mercury may range from 2 to 38 percent, and is nearly complete for organic mercury. Mercury accumulates primarily in the kidney. Elemental and organic mercury can also be transferred through the blood-brain barrier and the placenta because of their high lipophilicity. Inorganic mercury compounds can reach most organs, but accumulation in the brain and fetus is reduced because of their low lipophilicity. Following exposure to elemental mercury, excretion occurs via the urine, feces, and expired air. Following exposure to inorganic mercury, excretion occurs via the urine and feces. Organic mercury compounds are excreted primarily in the inorganic form via the urine and feces (ATSDR, 1988).

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MOLYBDENUM

(CAS RN 7439-98-7)

Molybdenum is a nutritionally essential trace metal in humans that is used as a cofactor for the enzymes xanthine oxidase and aldehyde oxidase. In plants it is necessary for fixing of atmospheric nitrogen by bacteria at the start of protein synthesis. Hence, it is ubiquitous in food. The average daily human intake of molybdenum in food is approximately 350 µg (Casarett and Doull, 1986).

The most important source of molybdenum is molybdenite (MoS_2). Industrial uses of this metal include the manufacture of high-temperature resistant steel alloys for use in gas turbines and jet aircraft engines, and the production of catalysts, lubricants, and dyes.

Absorption/Distribution/Metabolism/Excretion

Molybdenum from soluble compounds is readily absorbed from the GI or respiratory tracts (Friberg and Lener, 1986). Estimates of GI absorption in humans average around 50 percent, with a range of 38 to 72 percent observed in young women, and 77 percent reported for school children. The form of oxidation state of molybdenum used in these studies was not specified. Estimates of GI absorption in laboratory animals have ranged from 40 to 85 percent for hexavalent molybdenum.

Inhalation uptake studies with guinea pigs showed that molybdenum disulfide was essentially unabsorbed, but that hexavalent molybdenum was absorbed to an appreciable (unquantified) extent (Friberg and Lener, 1986). Absorbed molybdenum was distributed primarily to the kidneys, and bone in several animal models (Friberg and Lener, 1986). Molybdenum appears to accumulate in the liver, cartilage of the long bones, and skin. In humans and most animal models, the kidneys are the principal organs of excretion. The excretion of molybdenum is affected by the level of copper and sulfate in the diet.

Acute Toxicity

Chronic molybdenum poisoning in livestock has resulted from a molybdenum-copper imbalance and is characterized by anemia, GI disturbances, bone disorders, and growth depression (Friberg and Lener, 1986). In laboratory animals, excess molybdenum has induced effects in the liver, kidneys, and spleen. Gout-like symptoms were observed in humans living in a high molybdenum, low-copper area. A few cases of pneumoconiosis were reported in occupationally exposed workers. The EPA (IRIS, 2000) has derived a provisional chronic oral RfD of 5.0E-03 mg/kg-day, based on a LOAEL in humans exposed to high levels in water and diet, and on an uncertainty factor of 30. The critical effect associated with the oral RfD is increased uric acid levels. The UF of 30 results from a factor of 3 for sensitive human populations and a factor of 10 for use of a LOAEL rather than a NOAEL. Target organs for molybdenum toxicity include the erythrocyte, joints, liver, and kidneys.

Carcinogenicity

No studies were located regarding the human carcinogenicity of molybdenum.

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PHTHALATES

Di-n-octyl phthalate (DOP), bis (2-ethyl hexyl), phthalate (BEHP), and di-n-butyl phthalate (DBP), are members of the phthalate and phthalate esters family. Because of the lack of compound-specific information and the similarity of toxicity of the group of compounds, the general toxicity of the family of compounds with specific examples of various phthalate compounds will be presented.

Phthalate esters are used as plasticizers in almost every product category. They are used in construction, household products, toys, packaging, apparel, and medical products (Klaassen, et al., 1986).

Phthalates do not dissolve easily in water nor do they evaporate easily. However small amounts can enter the air as a gas. Phthalates once in the air generally break down within a few days. In soil, phthalates are broken down by the bacteria present which can take anywhere from a day up to a month depending on what types of bacteria are present and the temperature of the soil (Johnson, et al., 1977).

Phthalates appear to have relatively low toxicity. What animal data are available indicate that it takes consumption of large amounts to produce adverse effects, which are primarily reproductive.

Acute Effects

Acute lethality studies in animals indicate low acute toxicity of DOP, BEHP, and DBP. Study findings as observed by Smith (1953), Hardin, et al (1987), and White, et al. (1983) include single lethal doses of 8000 mg/kg up to more than 20,000 mg/kg.

Systemic Effects

An oral reference dose (RfD) of 2E-2 mg/kg/day for BEHP based on no observed effects as to relative liver weight is reported by the EPA (IRIS, 2000). An oral RfD of 1E-1 mg/kg/day is reported by the EPA for DBP. The BEHP value is derived from a study conducted by Carpenter, et al. (1953) in which rats and guinea pigs were exposed to BEHP at 20-195 g/kg day. A lowest-observed-adverse-effects level (LOAEL) was identified and converted to 19 mg/kg/day of BEHP.

Carcinogenic Effects

Sittig (1985) reports that no studies on the carcinogenic potential of DOP are available. BEHP however has been classified as B2 (probable human carcinogen), by the EPA on the basis of dose related increases in liver tumor responses in rats and mice, both male and female (IRIS, 2000; HEAST, 1990). One group of 100 rats and one of 100 mice each were fed 0, 6000, and 12,000 ppm BEHP for 103 weeks. The increase in hepatocellular carcinoma and combined incidence of carcinoma and adenoma were significant (NTP, 1982). These findings are supported by data by Ganning, et al. (1984) that has shown BEHP to be a potent inducer of hepatic peroxisomal enzyme activity. The oral slope factor for BEHP is 1.4E-2 (mg/kg/day)-1 which is equivalent to an oral carcinogenic unit risk of 4E-7 (µg/l)-1 (IRIS, 2000).

Teratogenicity, Embryotoxicity, And Reproductive Effects

Intraperitoneal injection of DOP in pregnant rats has caused teratogenic effects, increased resorptions and fetal toxicity (EPA, 1980). DBP has been shown to be toxic to the fetus (IRIS, 2000). BEHP has been observed to be fetotoxic and teratogenic also (Shiot and Nishimura, 1982).

The phthalates have also been associated with reproductive effects including seminiferous tubule damage and testicular damage (Shiota and Nichimura, 1982; Singhe, 1972; IRIS, 2000).

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POLYCYCLIC AROMATIC HYDROCARBONS

Polycyclic aromatic hydrocarbons (PAHs) are formed during the incomplete combustion of organic substances (e.g., coal, oil, gas, garbage). There are over 100 PAHs; among the most common are: acenaphthene, acenaphthylene, anthracene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, fluoranthene, fluorene, indeno(1,2,3-cd)pyrene, naphthalene, phenanthrene, and pyrene. The toxicity information available on PAHs is limited. Therefore, the potential health effects of "unknown" PAHs are inferred from those listed above. PAHs are generally found in the environment as mixtures of 2 or more compounds. Most PAHs are not readily soluble in water. Some easily volatilize into air; generally, PAHs will persist in the environment for months to years. PAHs are found throughout the environment. Background levels are 0.02 to 1.2 mg/m^3 (rural) to 0.15 to 19.3 mg/m^3 (urban) and 4 to 24 ng/L, for air and water respectively.

PAHs can be classified according to the electron density associated with the molecule as alternate or non-alternate. This distinction is toxicologically significant because the two classes appear to behave differently, particularly with respect to how the molecule is metabolized.

Absorption/Distribution/Metabolism/Excretion

PAHs appear to be readily bioavailable following inhalation exposure. Absorption of PAHs following inhalation has been inferred from the presence of urinary metabolites of PAHs in humans following exposure (Becher and Bjorseth, 1983 Cited in ATSDR, 1990). However, the dose-uptake relationship was not linear over the exposure concentration range, probably due to incomplete availability of the PAHs adsorbed to airborne particulate matter. Studies in animals suggest that clearance of PAHs from the lung is the product of direct absorption into the blood stream and clearance by mucociliary action and subsequent ingestion. The size of the particles to which the PAHs are adsorbed will influence the relative degree of absorption into the blood stream from the lungs (Sun et al., 1982; Cresia et al., 1976 cited in ATSDR, 1990). Benzo(a)pyrene was not bioavailable following direct nasal instillation in hamsters, monkeys, or

dogs; local mucosal absorption and metabolism were predominant (Dahl et al., 1985 and Petridou-Fischer et al., 1988 cited in ATSDR, 1990)

The oral bioavailability of PAHs is variable. Benzo(a)pyrene could not be detected in human feces following ingestion of broiled meat containing benzo(a)pyrene; blood levels did not appear to have been measured (Hecht et al., 1979 cited in ATSDR, 1990). Many of the animal studies only evaluated recovery of the administered dose in the feces and failed to measure blood or urine levels; therefore, a direct indication of bioavailability was not determined. In two studies, benzo(a)pyrene and benzo(a)anthracene were detected in the liver, lung, kidney, blood, and brain; however, no quantitative data were given (Yamazaki et al., 1987; Modica et al., 1982 cited in ATSDR, 1990).

PAHs are absorbed through the skin of both humans and animals. PAHs could be detected in the blood following application of crude coal tar to the skin of humans (Storer et al., 1984 cited in ATSDR, 1990). Three percent of an applied dose of ^{14}C-benzo(a)pyrene permeated human skin in vitro (Kao et al., 1985 cited in ATSDR, 1990). In mice, 93 percent of a dermally applied dose of ^{14}C-benzo(a)pyrene was recovered in the feces after 7 days, indicating that only about 7 percent was absorbed (Sanders et al., 1986 cited in ATSDR, 1990). Fifty-three percent of a dermally applied dose of ^{14}C-anthracene was recovered in the urine, feces, and tissues of rats over a 6 day period (Yang et al., 1986 cited in ATSDR, 1990). A number of other PAHs were absorbed following dermal exposure, but the amount absorbed is not linear with dose, suggesting saturation of an uptake process (Sanders et al., 1986 cited in ATSDR, 1990).

The distribution of PAHs in humans is unknown. In animals, benzo(a)pyrene has been predominant PAH studied. PAHs were more widely distributed following oral exposure than inhalation exposure; and, distribution was limited following dermal exposure. Typically, the highest distribution was to the lung, liver, kidney, and GI tract (Weyand and Bevan 1986, 1987, 19888; Schnizlein et al., 1987; Sun et al., 1982; Bartosek et al., 1984; Daniel et al., 1967 cited in ATSDR, 1990). Concentrations of PAHs were detected in the fetus following oral administration to the dam. However, the differences in fetal concentration appeared to reflect differences in GI

uptake in the dam and not ability to permeate the placenta (Shendrikova and Aleksandrov, 1974 cited in ATSDR, 1990). The majority of radioactivity was recovered in the lipid fraction of the tissues (Yamazaki et al., 1987 cited in ATSDR, 1990).

PAH metabolism has been studied extensively in vivo (animals) and in vitro (human and animal cells) (ATSDR, 1990). In general, metabolism transforms lipophilic PAHs into more water-soluble, excretable compounds. Because of the structural similarities among PAHs, benzo(a)pyrene has been used as a model for PAH metabolism. Benzo(a)pyrene is metabolized by the microsomal enzyme, aryl hydrocarbon hydroxylase (AHH). This enzyme is inducible in some species. The inducibility of this enzyme has been shown to be associated with increased survival time (Robinson et al. 1975 cited in ATSDR, 1990). PAHs also induce aldehyde dehydrogenase (ADH) activity with a variable potency that has been correlated with carcinogenic potency (Torronen et al., 1981 cited in ATSDR, 1990). Some PAHs are also able to induce carboxylesterase activity (Nousiainen et al., 1984 cited in ATSDR, 1990). Epoxide hydrolase metabolism of PAHs may lead to epoxide formation, increasing the likelihood for carcinogenic effects in organs with this enzyme activity (Moore et al., 1982 cited in ATSDR, 1990). There is some evidence to suggest that formation of bay-region diol epoxides are not the reactive intermediates associated with the tumorigenic effect of benzo(b)fluoranthene and other non-alternate PAHs (Amin et al., 1982, 1985 cited in ATSDR, 1990).

Studies on the elimination of PAHs are limited. However, PAHs and their metabolites have been recovered in variable amounts in the urine, feces, and/or bile after oral, dermal, or inhalation exposure (ATSDR, 1990). Following intravenous administration of ^{3}H-benzo(a)pyrene, the overall elimination was fit to a triexponential model; the final elimination half-life was 178 minutes (Weyand and Bevan, 1986 cited in ATSDR, 1990).

Acute Toxicity

A single exposure to PAHs has been associated with a number of toxic effects, including initiation of carcinogenicity (see target organ toxicity and carcinogenicity sections for details).

The acute lethal toxicity of PAHs ranges from 250 mg/kg for the most potent to 700 mg/kg for the least potent. However, most exposure situations will involve a mixture of PAHs; and, it is not possible to tell whether the acute lethal toxicity for a mixture will lie somewhere in between these values or whether toxicity will be additive. In addition, these data were obtained following parenteral administration, a step which by-passes metabolism in the liver; therefore, their relevance to human exposure is questionable.

Target Organ Toxicity

Pyrene (CASN 129-00-0). The EPA has established an oral RfD (RfD) of 3E-2 based on administration of 0, 75, 125, or 250 mg/kg/day pyrene by oral intubation to mice for 13 weeks (EPA, 2000 cited in IRIS). Minimal to mild nephropathy (renal tubular pathology and decreased kidney weights) was observed; the dose relationship was moderate. The UF was 3,000 due to species variability and lack of additional toxicity studies. The overall confidence in the RfD is low due to gaps in the database.

Fluoranthene (CASN 206-44-0). The EPA has established a RfD of 4E-2 based on gavage administration of 0, 125, 250, or 500 mg/kg/day fluoranthene to mice for 13 weeks (IRIS, 2000). The mid and high dose mice exhibited nephropathy, a dose-dependent increase in liver enzymes and weights, and increased salivation. The UF was 3,000 due to species variability, the use of a subchronic study, and lack of additional toxicity data. The overall confidence in the RfD is low due to gaps in the database.

Acenaphthene (CASN 83-32-9). The EPA has established an oral RfD of 6.0E-2 for acenaphthene (IRIS, 2000) based on hepatotoxicity during an oral subchronic study on mice. The UF of 3,000 was due to species variability, use of a subchronic study, lack of adequate data in a second species, and lack of reproductive/developmental data.

Anthracene (CASN 120-12-7). The EPA has established an oral RfD of 3.0E-01 for anthracene (IRIS, 2000) based on no observed effects in a subchronic study on mice. The UF of 3,000 was due to species variability, use of a subchronic study, lack of adequate data in a second species, and lack of reproductive/developmental data.

Naphthalene (CASN 91-20-3). The EPA has established an oral RfD of 2.0E-02 for naphthalene (IRIS, 2000) based on no observed effects in a subchronic study on rats. The UF of 3,000 was due to species variability, use of a subchronic study, lack of adequate data in a second species, and lack of reproductive/developmental data.

GI Toxicity. Carboxylesterase activity of the intestinal mucosa was decreased in rats following intragastric administration of 50 or 150 mg/kg/day benzo(a)anthracene or benzo(a)pyrene for 4 days; enzyme activity was increased following oral exposure to 100 mg/kg/day of anthracene or phenanthrene (Nousiainen et al., 1984 cited in ATSDR, 1990).

Hematological Toxicity. Bone marrow depression (aplastic anemia and pancytopenia) leading to death was observed in mice whose AHH enzyme was not inducible following oral administration of 120 mg benzo(a)pyrene/kg/day for 180 days (Robinison et al., 1975 cited in ATSDR, 1990).

Hepatic Toxicity. A number of hepatic effects (enzyme and foci induction, liver regeneration, and increased weights) have been observed in animals administered PAHs. These effects are not life-threatening but may precede the onset of more serious effects. A single intragastric administration of 200 mg/kg of benzo(a)pyrene, benzo(a)anthracene, or dibenz(a,h)anthracene induced the formation of preneoplastic hepatocytes in a promotion/initiation bioassay in partially hepatectomized rats fed 2-acetylaminofluorene (Tsuda and Farber, 1980 cited in ATSDR, 1990). PAHs stimulate liver regeneration in partially hepatectomized rats following administration in the diet for 10 days. Non-carcinogenic PAHs required higher doses (Gershbein, 1975 cited in ATSDR, 1990).

Dermal Toxicity. Dermal exposure to benzo(a)pyrene in humans was associated with epidermal changes indicative of neoplastic proliferation, chronic dermatitis and hyperkeratosis, or exacerbation of pre-existing skin lesions (Cottini and Mazzone, 1939; EPA, 1988 cited in ATSDR, 1990). Topical application of PAHs have been associated with adverse effects in animals including skin cancer, effects on sebaceous glands, increased number of skin melanocytes, dermal inflammation, allergic contact hypersensitivity, and photosensitization (Bock and Mund, 1958; Iwata et al., 1981 Klemme et al., 1987; Old et al., 1963 and Forbes et al., 1976 cited in ATSDR, 1990).

Immunotoxicity Immunotoxic effects have been observed following dermal and parenteral administration in animals. PAHs that are carcinogenic are also immunosuppressive with the same rank order of potency (ATSDR, 1990). Effects observed have included inhibition of T-cell dependent and independent antibody production and inhibition of lymphocyte mediated immunity (Blanton et al., 1986; Lyte and Bick, 1985; Whit and Holsapple, 1984; Wojdani et al., 1984 cited in ATSDR, 1990).

Developmental Toxicity

There is no information on the developmental effects of exposure to PAHs in humans (ATSDR, 1990)

In animals, in utero exposure to benzo(a)pyrene (10, 40, or 160 mg/kg/day orally during gestation) was associated with reduced mean postnatal pup weight and increased incidence of sterility associated with alterations in gonadal morphology and germ-cell development in male mice (Mackenzie and Angevine, 1981 cited in ATSDR, 1990). An observed increased incidence of stillborns, resorptions, and malformations following dietary administration of 120 mg benzo(a)pyrene/kg/day to the dam was associated with the metabolic responsiveness of the offspring and the dams (Legraverend et al., 1984 cited in ATSDR, 1990) Parental administration of a number of PAHs has been associated with a number of developmental effects: stillbirths,

increased fetal resorptions, malformations, adverse gonadal histology, and decreased fetal survival. The implications of the results obtained following parenteral administration, which by-passes first-pass metabolism of the liver, to human exposure are that adverse effects on the fetus are possible.

Reproductive Toxicity

There is no information on the effect of PAH on reproduction in humans (ATSDR, 1990).

Benzo(a)pyrene decreased the percentage of pregnant females at parturition in pregnant CD-1 mice (Mackenzie and Angevine, 1981 cited in ATSDR, 1990) and reduced the incidence of pregnancy in rats (Rigdon and Rennels, 1964 cited in ATSDR), but it had no effect on fertility of Swiss mice (Rigdon and Neal, 1965 cited in ATSDR, 1990). Increased resorptions and decreased number of corpora lutea, uterine weights, and fetal survival were observed following parenteral administration of benzo(a)pyrene (Swartz and Mattison, 1985; Bui et al , 1986 cited in ATSDR, 1990). The implications of the results obtained following parenteral administration, which by-passes first-pass metabolism of the liver, to human exposure are that adverse effects on reproduction are possible.

Genotoxicity

A number of PAHs have been tested in numerous in vitro and in vivo genotoxicity studies (ATSDR, 1990). In general, several PAHs demonstrated genotoxic potential that required metabolic activation. Benzo(a)pyrene produced several genotoxic effects: DNA binding/damage, sister chromatic exchange, chromosomal aberration, cell transformation; effects were observed in bacterial and mammalian (both somatic and germ) cells. DNA damage was also observed in human cells. Acenaphthene, acenaphthylene, or fluorene were negative for genotoxic effects and anthracene, phenanthrene, and pyrene where negative in all but one in vitro test.

Carcinogenicity

Epidemiologic studies of humans exposed to coke-oven and roofing-tar emissions and cigarette smoke have demonstrated an increase in mortality due to lung cancer (Lloyd, 1971; Mazumdar et al., 1975; Redmond et al., 1976; Hammond et al., 1976; cited in ATSDR, 1990). Skin tumors have been reported among individuals exposed to mixtures containing PAHs: scrotal cancer among chimney sweeps and skin cancer following exposure to shade oils (Pott, 1775; Purde and Etlin, 1980 cited in ATSDR, 1990). While these mixtures contain PAHs they also contain a number of other potentially carcinogenic chemicals, tumor promoters, initiators, and cocarcinogens. This concomitant exposure prevents making a direct causal relationship to PAHs.

Inhalation, oral, and dermal exposure to PAHs have been associated with carcinogenic effects in animals (ATSDR, 1990). These studies suggest that the site of tumor induction is influenced by the route of administration. Following inhalation exposure an increased incidence of respiratory tract tumors was observed. Benign and malignant tumors, adenomas, papillomas, neoplasms, and carcinomas of the lung, alimentary tract (particularly the forestomach), and mammary glands were observed following ingestion of benzo(a)pyrene, benzo(a)anthracene and dibenz(a,h)anthracene by rodents. A dose-responsive increase in the incidence of forestomach papillomas and carcinomas was observed in mice following dietary administration of 33.3 mg benzo(a)pyrene/kg/day for 30 to 197 days (Neal and Rigdon, 1967 cited in ATSDR, 1990). While the relevance of forestomach tumors in rodents to human cancer is debatable and not all PAHs are associated with a carcinogenic or genotoxic effect, the overall evidence suggests exposures to PAHs are associated with an increased risk of cancer.

Benzo(a)anthracene, benzo(b)fluoranthene, benzo(a)pyrene, dibenz(a,h)anthracene, chrysene, and indeno(1,2,3-cd)pyrene have been demonstrated to induce skin tumors in mice following dermal exposure (ATSDR, 1990). Benzo(a)pyrene, in particular, is a potent experimental skin carcinogen. It is often used as a positive control in carcinogenesis bioassays. The dose-response relationship of benzo(a)pyrene and skin tumors has been investigated in a number of studies (Cavalieri et al., 1988; Shubik and Porta, 1957; Bingham and Falk, 1969; Wynder and Hoffman,

1959; Warshawsky and Barkley, 1987 cited in ATSDR, 1990) Benzo(a)pyrene has been demonstrated to induce malignant skin tumor at exposures as low as 0.0054 mg/kg/day; however, the solvent and strain of mouse tested will influence the tumorigenic dose (Bingham and Falk, 1969; Warshawsky and Barkley, 1987 cited in ATSDR, 1990). The latency of tumor production by benzo(a)pyrene may also be affected by the schedule of administration because of the ability of this PAH to induce the enzyme responsible for its metabolism (Slaga and diGiovanni, 1984; Weibel, 1980 cited in ATSDR, 1990).

Pyrene (CASN 129-00-0). Mice administered intraperitoneal injections of pyrene on days 1, 8, and 15 of age did not demonstrate a treatment-related increase in tumor incidence (Wislocki et al , 1986 cited in IRIS, 2000). Pyrene was not positive or inconclusive in mouse skin-painting assays (Badger et al., 1940; Horton and Christian, 1974; Van Duuren and Goldschmidt, 1976; Salaman and Roe, 1956; Scribner, 1973 cited in IRIS, 2000). Subcutaneous injection of pyrene did not produce tumors in Jackson A mice (Shear and Leiter, 1941 cited in IRIS, 2000). The EPA has classified pyrene as a class D carcinogen; not classifiable as to human carcinogenicity due to no human data and inadequate data from animal bioassays.

Benzo(a)pyrene (CASN 50-32-8). There are multiple animal studies demonstrating increased incidence of carcinogenic effects following oral, intratracheal, inhalation, and dermal administration of benzo(a)pyrene Benzo(a)pyrene has been shown to be an initiator and a complete carcinogen following dermal application (IARC, 1973 cited in IRIS, 2000). A dose-responsive increase in squamous cell papillomas and carcinomas of the forestomach was observed following oral administration of 1 to 250 ppm benzo(a)pyrene to rats and hamsters for 197 days (Rigdon and Neal, 1966, 1969 cited in IRIS, 2000). The incidences of respiratory tract and upper digestive tract tumors were increased following intratracheal instillation or inhalation of benzo(a)pyrene by guinea pigs, hamsters, and rats (EPA, 1991 cited in IRIS, 2000). Inhalation exposure of hamsters to benzo(a)pyrene at 9.5 mg/cubic m/day for 10 weeks was associated with development of tumors of the nasal cavity, larynx, trachea, and pharynx. At the next highest dose, 45 mg/m^3/day, neoplasms were also observed in the upper digestive tract. The lowest dosed (2.2 mg/m^3/day) animals did not develop tumors (Thyssen et al., 1981 cited in IRIS, 2000).

Intraperitoneal and subcutaneous injection of benzo(a)pyrene is associated with injection site tumors (EPA, 1991 cited in IRIS, 2000) Based on no human data and sufficient animal data benzo(a)pyrene is classified, B2; probable human carcinogen. EPA has calculated an oral slope factor of 7.3E+00 $(mg/kg/day)^{-1}$, and a corresponding drinking water unit risk of 2.1E-04 (ug/L) (IRIS, 2000). Cal EPA has established an oral slope factor of 1.2E+01 $(mg/kg/day)^{-1}$, and an inhalation slope factor of 3.9E+00 (Cal EPA, 1994). The inhalation slope factor corresponds to a unit risk value of 1.1E-03 $(ug/m^3)^{-1}$ (Cal EPA, 1994).

Benzo(k)fluoranthene (CASN 207-08-9). In a lifetime study in female rats, lung implants of 0.65, 3.4, or 17 mg/kg benzo(k)fluoranthene exhibited a dose-related increase in the incidence of epidermoid carcinomas in the lung and thorax. Equivocal incidences of lung adenomas and hepatic adenomas and hepatomas were reported in mice administered intraperitoneal injections of 120 µg benzo(k)fluoranthene/mouse on days 1, 8, and 15 of age (LaVoie et al., 1987 cited in IRIS, 2000). Benzo(k)fluoranthene was positive in mouse skin-painting assays (Van Duuren et al., 1966; LaVoie et al , 1982; Amin et al., 1985 cited in IRIS, 2000). Based on these data, EPA has classified benzo(k)fluoranthene as B2 "probable" human carcinogen. Using a potency equivalency factor (PEF) of 0.1, the benzo(a)pyrene inhalation slope factor of 3.9E+00 $(mg/kg/day)^{-1}$, and the oral slope factor of 1.2E+01, Cal EPA has established an inhalation slope factor of 3.9E-01 $(mg/kg/day)^{-1}$, and an oral slope factor of 1.2E+00 for benzo(k)fluoranthene (Cal EPA, 1994).

Indeno(1,2,3-cd)pyrene (CASN 193-39-5). EPA has classified indeno(1,2,3-cd)pyrene as a B2 probable human carcinogen (IRIS, 2000). Using a PEF of 0.1, the benzo(a)pyrene inhalation slope factor of 3.9E+00 $(mg/kg/day)^{-1}$, and the oral slope factor of 1.2E+01, Cal EPA has established an inhalation slope factor of 3.9E-01 $(mg/kg/day)^{-1}$, and an oral slope factor of 1.2E+00 for indeno(1,2,3-cd)pyrene (Cal EPA, 1994).

Benzo(b)fluoranthene (CASN 205-99-2). In a lifetime study in female rats, lung implants of 0.4, 1.2, or 4.1 mg/kg benzo(b)fluoranthene exhibited a dose-related increase in the incidence of epidermoid carcinomas and pleomorphic sarcoma in the lung and thorax. Equivocal incidences

of lung adenomas and hepatic adenomas and hepatomas were reported in mice administered intraperitoneal injections of 126 µg benzo(b)fluoranthene/mouse on days 1, 8, and 15 of age (LaVoie et al., 1987 cited in IRIS, 2000). Injection site sarcomas were observed in mice administered subcutaneous injections of benzo(b)fluoranthene (2.6 mg total dose) over 2 months (Lacassagne et al., 1963 cited in ATSDR, 1990). Benzo(b)fluoranthene was positive for complete carcinogenesis and initiation in mouse skin-painting assays (Wynder and Hoffmann, 1959; LaVoie et al., 1982; Amin et al., 1985 cited in IRIS, 2000). Based on these data, EPA has classified benzo(k)fluoranthene as B2 "probable" human carcinogen. Using a PEF of 0.1, the benzo(a)pyrene inhalation slope factor of 3.9E+00 $(mg/kg/day)^{-1}$, and the oral slope factor of 1.2E+01, Cal EPA has established an inhalation slope factor of 3.9E-01 $(mg/kg/day)^{-1}$, and an oral slope factor of 1.2E+00 for benzo(b)fluoranthene (Cal EPA, 1994).

Dibenz(a,h)anthracene (CASN 53-70-3). U.S. EPA has calculated an oral slope factor of 7.3E+00 $(mg/kg/day)^{-1}$ for dibenz(a,h)anthracene, based on its relative toxicity to benzo(a)pyrene. Dibenz(a,h)anthracene has been classified as a B2 probable human carcinogen. Cal EPA has established an oral and inhalation slope factor of 4.1 $(mg/kg/day)^{-1}$ (Cal EPA, 1994) The inhalation slope factor corresponds to a unit risk value of 1.2E-03 $(ug/m^3)^{-1}$ (Cal EPA, 1994).

Acenaphthylene (CASN 208-96-8). Dermal application of acenaphthylene (0.25 percent) did not cause tumor development in a lifetime study in mice (Cook, 1932). While no control was used in this study, other PAHs tested did result in skin tumor formation EPA has classified acenaphthylene as a class D carcinogen; not classifiable as to human carcinogenicity (IRIS, 2000).

Anthracene (CASN 120-12-7). EPA has listed anthracene as a class D carcinogen; not classifiable as to human carcinogenicity due to lack of human data and inadequate animal bioassay data (IRIS, 2000).

Phenanthrene (CASN 85-01-8). Two skin painting assays were negative for complete carcinogenic activity; 1 out of 5 mouse skin painting assays was positive for initiation (IRIS, 2000). Tumorigenic activity was not detected following intraperitoneal injection of phenanthrene (0.25 mg total dose) on days 1, 8, and 15 after birth (Buening et al , 1979 cited in IRIS, 2000). A single subcutaneous injection of 40 µg phenanthrene in albino mice was not overtly tumorigenic (Grant and Roe, 1963 cited in IRIS, 2000). No tumors were reported in mice receiving a single subcutaneous injection of 5 mg phenanthrene (Steiner, 1955 cited in IRIS, 2000) EPA has classified phenanthrene as a class D carcinogen; not classifiable as to human carcinogenicity.

Benzo(a)anthracene (CASN 56-55-3). Increased incidence of pulmonary tumors and hepatomas was observed in mice following oral gavage with 500 mg benzo(a)anthracene/kg/day 3 times/week for 5 weeks (Klein, 1963 cited in IRIS, 2000). A single gavage dose of 0.5 mg benzo(a)anthracene was not tumorigenic, while multiple gavage treatments (8 or 16 over 16 months) resulted in forestomach papillomas (Bock and King, 1959 cited in IRIS, 2000). Following intraperitoneal injection of benzo(a)anthracene on days 1, 8, and 15 of age (638 µg/mouse total dose) the incidence of liver adenomas or carcinomas was increased in treated male mice, while the incidence of pulmonary adenomas or carcinomas was increased in treated females (Wislocki et al., 1986 IRIS, 2000). Benzo(a)anthracene was positive as a complete carcinogen and as an initiator in mouse skin painting bioassays (IARC, 1973 cited in IRIS, 2000). Injection site sarcomas were observed following subcutaneous or intramuscular injection of benzo(a)anthracene (Steiner and Falk, 1951; Steiner and Edgecomb, 1952; Klein, 1952 cited in IRIS, 2000). EPA has classified benzo(a)anthracene as a B2 "probable" human carcinogen. Using a PEF of 0.1, the benzo(a)pyrene inhalation slope factor of 3.9E+00 $(mg/kg/day)^{-1}$, and the oral slope factor of 1.2E+01, Cal EPA has established an inhalation slope factor of 3.9E-01 $(mg/kg/day)^{-1}$, and an oral slope factor of 1.2E+00 for benzo(a)anthracene (Cal EPA, 1994).

Naphthalene (CASN 91-20-3). No treatment-related tumors developed in rats fed a diet supplemented with 360 mg naphthalene/kg/day (total dose administered was 10 g/rat) (Schmahl, 1955 cited in IRIS, 2/93). Pulmonary tumors developed in mice following inhalation exposure to

0, 10, or 30 ppm naphthalene for 6 hours/day, 5 days/week for 6 months; no dose-response was observed (Adkins et al., 1986 cited in IRIS, 2000) The number of gamma-glutamyl transpeptidase positive foci, an early neoplastic indicator, was not increased following single gavage administration of 100 mg naphthalene/kg (Tsuda et al., 1980 cited in IRIS, 2000). No carcinogenic responses were observed in rats after intraperitoneal injection of 20 mg naphthalene/kg once a week for 40 weeks (Schmahl, 1955 cited in IRIS). Naphthalene was negative as a complete carcinogen or initiator in mouse skin-painting assays (Kennaway, 1930; Schmelts et al., 1978 cited in IRIS) EPA has classified naphthalene as a class D carcinogen.

Benzo(g,h,i)perylene (CASN 191-24-2). Benzo(g,h,i)perylene was negative as a complete carcinogen or an initiator in mouse skin painting assays (IRIS, 2000). Rats with benzo(g,h,i)perylene lung implants did not develop increased incidence of epidermoid carcinomas in the lung or thorax (Deutsch-Wenzel et al., 1983). Mice injected subcutaneously with 0.1, 1, or 10 mg benzo(g,h,i)perylene once every 2 weeks for 20 weeks did not develop tumors (Muller, 1968 cited in IRIS, 2000). EPA has classified benzo(g,h,i)perylene as a class D carcinogen.

Chrysene (CASN 218-01-9). Chrysene tested positive as a complete carcinogen and an initiator in mouse skin painting studies (IRIS, 2000). The incidence of liver adenomas or carcinomas and lung adenomas was increased in male, but not female, mice administered intraperitoneal injections of chrysene on days 1, 8, and 15 of age (total dose was 160 or 640 µg/mouse) (Wislocki et al., 1986 cited in IRIS, 2/93). Intraperitoneal injection of chrysene on days 1, 8, and 15 of birth resulted in increased incidence of hepatic tumors in treated male mice and not treated females (Chang et al., 1983). Using a PEF of 0.01, the benzo(a)pyrene inhalation slope factor of 3.9E+00 $(mg/kg/day)^{-1}$, and the oral slope factor of 1.2E+01, Cal EPA has established an inhalation slope factor of 3.9E-02 $(mg/kg/day)^{-1}$, and an oral slope factor of 1.2E-01 for chrysene (Cal EPA, 1994).

Fluoranthene (CASN 206-44-0). Fluoranthene was consistently negative as a complete carcinogen in mouse skin painting studies (IRIS, 2000). EPA has classified fluoranthene as a class D carcinogen.

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SELENIUM

(CAS RN 7782-49-2)

Selenium toxicity depends on total body levels since its deficiency and excess result in several disorders (ASTDR, 1989). The nature of the toxicity does not appear to correlate with the oxidation state of selenium but absorption and bio-availability depend on the form of selenium (i.e., organic or inorganic).

Acute Toxicity

Low body and tissue levels of selenium result in diminished glutathione peroxidase enzyme activity in whole blood and erythrocytes which adversely effects the protection of cells from oxidation damage (Valentine et. al, 1988). Depressed selenium levels result in at least two chronic, non-cancerous, metabolic diseases: Keshan disease and Kashin-Beck disease (Yang et. al, 1988). Keshan disease manifests its symptoms by increased necrosis of the myocardial muscle, while Kashin-Beck disease results in degeneration, atrophy, and necrosis of cartilage. Low selenium intake in other instances results in increased cardiomyopathy resulting in increased cardiovascular deaths in man (Oster et. al, 1983; Salonen et. al, 1982).

Excessive body levels of selenium manifest their symptoms as increased garlic breath, increased skin rashes, dental carie increase, brittle and discolored nails, hair loss and increased nervous system disorders (Kilness and Hochberg, 1977; Yang et. al, 1983) Severe selenosis can result in chronic nervous system degeneration and depression.

In rodent species, oral ingestion of selenium and selenium salts produce acute toxicity to mice, rats, guinea pigs, and rabbits (ASTDR, 1989. Nonlethal doses of selenium sulfide or selenium disulfide result in pulmonary edema and respiratory congestion (Carter, 1966; Koppel et. at, 1986). Ingestion of high levels of selenacious plants results in respiratory failure in livestock (NAS, 1976a).

Severe gastrointestinal distress, abdominal cramping and fluid imbalance result from acute dosage with selenium salt in humans (Koppel, 1986). Livestock suffering from "blind staggers" exhibit severe gastrointestinal distress and upon necropsy, pronounced gastrointestinal necrosis (Shamberger, 1986). Gastrointestinal disorders in rodents fed grains high in selenium resulted in NOAELs of 0.5 mg/kg/day (ASTDR, 1989).

Chronic Toxicity

In chronic exposure settings with mice, myocardial amyloidosis of the heart was observed but the significance of this finding is unclear. Oral dosage of mice and rats with selenium salts results in myocardial hyperemia, hemorrhage, and degeneration, and pericardial edema (Schroeder and Mitchener, 1972).

Limited investigations in humans suggest that hepatic effects occur following chronic ingestion of selenium compounds. Abnormal liver function (serum bilirubin and alkaline phosphatase activity) has been demonstrated in one patient (Civil and MacDonald, 1978).

Hepatic congestion in ruminants and rodents have been noted (Hopper et. al, 1985; Palmer and Olsen, 1974). Chronic exposure to selenium compounds in rodents also results in depressed liver weight and eventually cirrhosis (NOAEL 0.68 mg/kg/day) (Harr et al, 1967)

Selenium intoxication has resulted in effects on other organ systems in rodents and humans. Renal effects such as nephritis and severe interstitial nephritis and any lordosis have been noted following oral ingestion of selenium compounds (Harr et. al, 1967). Oral ingestion of selenium has been associated with delayed eye lid development in mice (Ostadalova and Babicky, 1980).

The EPA (IRIS) lists a chronic oral reference dose RfD of 5E-03 (mg/kg/day) (IRIS, 2000).

Developmental Toxicity

There have been no investigations demonstrating that selenium or selenacious containing plants induce developmental anomalies in humans (ASTDR, 1989). Selenium does not induce terata in rodent species under very stringent conditions (Barlow and Sullivan, 1982). Avian species appear to be highly susceptible to selenium induced terata (Palmer et. at, 1973). Selenium ingestion elicits decreased fetal body weights in rats and mice and impairs fertility and conception rates in rodents (Chowdhury and Venkatakrishna-Ghatt, 1983). Selenium also decreases fecundacy in swine (Wahlstrom and Olson, 1959b). Recent investigations in primates indicate that selenium does not induce fetal malformations under continuous dosing prior to conception through parturition (Tarental et al, in press).

Genotoxocity

Selenium by itself has not been shown mutagenic in bacterial or mammalian cell mutagenesis assays (ASTDR, 1989). In fact, the presence of selenium in the mutation assays as a media supplement or as an adjunct to the S9 metabolic activation system decrease mutegenic activity of known mutagens (Gairola and Chow, 1982).

Carcinogenicity

Carcinogenicity data in rodents for dietary selenium present conflicting results. Early reports indicate that sodium selenate or selenacious plant material enhanced hepatic tumor incidence in rodents (Volgarev and Tscherkes, 1967; Schroder and Mitchener 1971a). Later reports indicate that dietary selenium inhibits spontaneous tumor development or chemically induced (N-2-fluorenylacetamide) tumors (Harr et.al, 1967). Selenium and selenium containing plants do not enhance the development of tumors in humans (ASTDR, 1989). In fact, in areas where high indigenous selenium occurs in the diet, tumor rates for tongue, esophagus, stomach, intestine, rectum, liver, pancreas, lung and bladder are significantly lower in males and females than in low selenium exposure diets (Shamberger et at, 1976). The possible mechanism may relate to effects on glutathione peroxidase levels (Valentine et. al, 1988).

Selenium sulfide is the only compound known to consistently increase tumor incidence in the form of hepatocellular carcinomas and alveolar/bronchiolar carcinomas in rats and mice (NTP, 1980c). Selenium sulfide is only used in topical pharmaceutical preparations and not taken orally. Lastly, in 1975, IARC concluded from the literature that selenium in the forms of selenite, selenate, and organic forms was non-carcinogenic to rodents and humans and is not considered to be a carcinogen.

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TETRACHLOROETHENE

(CAS RN 127-18-4)

Tetrachloroethene (PCE) is a man-made substance widely used for dry cleaning fabrics and textiles and for metal-degreasing operations. It is also used as a starting material for making other chemicals and is used in some consumer products. PCE enters the environment mostly by evaporating into the air during use, but it can also enter the environment by migrating from disposed sewage sludge or factory waste, and by leaching or evaporating from storage and waste sites.

Absorption/Distribution/Metabolism/Excretion

PCE is rapidly and completely absorbed following inhalation or ingestion by both humans and animals. In humans pulmonary uptake of PCE is proportional to ventilation rate, duration of exposure, and (at lower atmospheric concentrations of PCE) to the concentrations of PCE in the inspired air (Hake and Stewart, 1977 cited in ATSDR, 1990)

By contrast, dermal absorption of vapors of PCE in both humans and animals is insignificant. In human subjects fitted with a full facepiece respirator to prevent inhalation, only 1.0 percent of a dose that would have been expected to be inhaled was absorbed through the skin (Riihimake and Pfaffli, 1978 cited in ATSDR, 1990). An in vivo dermal absorption rate of 0.24 mg/cm^2/h in mice and an in vitro dermal absorption rate of 0.0055 mg/cm^2/h in rats has been reported (Tsuruta, 1975 cited in ATSDR, 1990). Regardless of the route of exposure, most of the absorbed PCE is excreted unchanged in exhaled air.

Once absorbed, physiologically based pharmacokinetic models indicate that PCE is largely distributed to fat tissue (Guberan and Fernandez, 1974 cited in ATSDR, 1990). This has been confirmed by experimental observation in animals. Following exposure of rats to 200 ppm PCE vapor for 5 days, PCE was distributed primarily to adipose tissue, especially the perirenal fat (Savolainen et al , 1977 cited in ATSDR, 1990); and, following gavage administration of PCE to

pigs, PCE was concentrated mainly in subcutaneous fat (Vemmer et al , 1984 cited in ATSDR, 1990) There is also evidence that once absorbed, PCE is able to cross the placenta and distribute to the fetus and amniotic fluid (Ghantous et al., 1986 cited in ATSDR, 1990).

As noted by Travis et al (1989), the metabolic pathways of PCE are still speculative, but there is convincing evidence that the principal site of metabolism is the hepatic microsomal cytochrome P-450 system. The first product of this system is thought to be a highly reactive, epoxide intermediate, 1,1,2,2-tetrachloroethene oxide, which then rearranges to form PCE's most prominent metabolite, trichloroacetic acid (TCA). There is evidence that the epoxide intermediate binds to cellular macromolecules. Both the epoxide and the ultimate metabolite, TCA, have been indicated as putative carcinogenic moieties (Travis et al , 1989). This metabolic pathway has been shown to be saturable This means that the rate of production of these toxic metabolites is limited by the activity of the metabolizing enzymes

In addition to this primary TCA pathway, PCE is believed to be metabolized also by secondary pathways that produce other principal metabolites, oxalic acid and CO_2 (Travis et al , 1989). These pathways are not believed to be saturable.

Acute Toxicity

Acute inhalation exposure to levels of PCE above 100 to 200 ppm in humans results in eye and upper respiratory irritation, headache, dizziness, and drowsiness (ATSDR, 1990). An inhalation LC_{50} value of 5,200 ppm PCE for mice and oral LD_{50} values of 3,835 and 3,005 mg/kg for male and female rats, respectively, have been reported (Friberg et al., 1953; Hazen et al., 1986 cited in ATSDR, 1990) Longer exposures to oral doses above 1,000 mg/kg 5 days/week result in death in rats (NCI, 1977 cited in ATSDR, 1990). Decreased longevity in mice and rats results from chronic exposures to about 471 mg/kg-day or 300 mg/kg-day, respectively, for 78 weeks (NCI, 1977 cited in ATSDR, 1990).

Target Organ Toxicity

The brain, liver ad kidney have been identified as target organs in humans for adverse noncarcinogenic effects of PCE exposure. The major types of noncarcinogenic effects are summarized by target organ below.

The EPA has derived an oral chronic reference for PCE of 1.0E-02 0.01 mg/kg-day based on the occurrences of hepatotoxicity in mice and weight gain in rats exposed to PCE in corn oil via gavage for six weeks (IRIS, 6/94). The UF of 1000 results from factors of 10 for each of the following: intraspecies variability, interspecies variability, and extrapolation to a chronic equivalent.

Neurotoxicity. Short inhalation exposures to PCE levels ranging from about 100 to 200 to 1,060 ppm result in dizziness, sleepiness, headache, and difficulty in speaking in humans; and cause ataxia and somnolence in rats (Rowe et al., 1952; Stewart et al., 1970; Carpenter, 1937; Goldberg et al., 1964 cited in ATSDR, 1990). Short oral exposures to doses ranging from 4 to 16 g cause similar effects (ATSDR, 1990). Animal studies of gerbils exposed for 3 or 12 months to PCE levels ranging from 60 to 120 ppm have demonstrated DNA, amino acid, and phospholipid alterations in the brain (Briving et al., 1986; Kyrkland et al., 1984; Rosengren et al., 1986 cited in ATSDR, 1990).

Hepatic Toxicity. PCE is a hepatotoxin in humans and animals by the inhalation and oral routes of exposure. The types of PCE-induced hepatic effects are reasonably well documented, but the exposures or doses producing these effects are less adequately characterized. As noted in ATSDR (1990b), for humans, reports of hepatotoxicity consist entirely of case studies of accidental exposures in which reliable quantitative exposure information was not available. Hepatotoxic effects reported include cirrhosis of the liver, toxic hepatitis, liver cell necrosis, hepatomegaly, and altered liver function indices (EPA, 1985 cited in ATSDR, 1990). In most cases, hepatic effects in humans have been reported as transient in nature.

In animals, interpretation of hepatotoxicity is complicated by differences in exposure schedule, sensitivity of end points, sensitivity of species, and a paucity of chronic-duration exposures. Hepatotoxic effects reported include fatty degeneration of the liver and increased liver weight, and pathological alterations in mice exposed to 200 ppm PCE for 4 hours or 9 ppm PCE for 30 days, respectively (Kylin et al., 1965; Kjellstrand et al., 1984 cited in ATSDR, 1990).

Mice are considered to be particularly sensitive to hepatotoxicity resulting from PCE exposure. Recent data indicate that short-term exposure to PCE by gavage or by inhalation induces peroxisome proliferation in mouse liver. Peroxisome proliferation has been suggested as a causative factor for the carcinogenic response in mouse liver (Goldworthy and Popp, 1987; Odum et al., 1988 cited in ATSDR, 1990). Experimental data also suggest that similar hepatic effects may be produced by either intermittent or continuous exposures when the time-weighted average dose is the same, and that PCE induced hepatic effects of intermediate-duration exposures are not totally reversible (ATSDR, 1990).

Renal Toxicity. Renal effects resulting from inhalation or oral exposure to PCE are documented in rodents but not in humans (ATSDR, 1990). However, PCE should be regarded as a probable nephrotoxin in humans because nonproliferative kidney lesions are characteristic effects of various halogenated aliphatic hydrocarbons (particularly chlorinated ethanes and ethylenes) which are structurally similar to PCE in animals and humans (Kluwe et al., 1984; NTP, 1986 cited in ATSDR, 1990).

Tetrachloroethene induces protein droplet nephropathy in male rats, which is characterized by accumulation of gamma-2u globulin in lysosomes, degeneration and necrosis of tubular cells, formation of granular casts, and regeneration of the tubular epithelium (Swenberg et al., 1988 cited in ATSDR, 1990). Chemicals that are known to induce protein droplet nephropathy bind to gamma-2u globulin, yielding a complex that is more resistant to the proteolytic enzymes in the lysosomes, leading to the accumulation of the complex in the tubule cells and subsequent nephropathy. Gamma-2u globulin has not been found in immature male rats, female rats, or humans (Alden, 1986 cited in ATSDR, 1990). If PCE induces nephropathy by the suggested

mechanism, the absence of gamma-2u globulin in humans raises the question of the relevance to humans of the PCE-induced kidney lesions in male rats

Cardiotoxicity. There are few reported cases of PCE-associated cardiotoxicity. A case study describing a 24-year old male who experienced skipped heart beats within one month after starting work in a dry-cleaning operation suggests an association of PCE with effects on the heart, but the man described may have been an unusually sensitive individual (Abeden et al , 1980 cited in ATSDR, 1990). It was hypothesized that exposure to PCE may have sensitized the myocardium to endogenous epinephrine. In an experiment in dogs, however, inhalation exposure to high levels of PCE failed to sensitize the heart to epinephrine (Reinhardt et al., 1973 cited in ATSDR, 1990). In contrast, intravenous administration of PCE enhanced myocardial sensitivity to an exogenous epinephrine challenge (Kobayashi et al., 1982 cited in ATSDR, 1990).

Immunotoxicity. Exposure of mice to 50 ppm PCE for 3 hours increased their susceptibility to bacterial respiratory infection (Aranyi et al., 1986 cited in ATSDR, 1990), suggesting that PCE may have an immunotoxic effect. There are no data on the potential immunotoxicity of PCE in humans.

Developmental Toxicity

Developmental studies have been conducted in rats and mice (one study) exposed to PCE by inhalation. The results of these studies indicate that PCE is fetotoxic, but not teratogenic, at concentrations that are also maternally toxic (ATSDR, 1990). This means that developmental toxicity resulting from direct effects of PCE is not easily discernible from developmental toxicity resulting from changes occurring in nature. Fetotoxicity was usually expressed by decreased fetal weight and delayed skeletal ossification. These effects have been associated with exposures greater than or equal to 300 ppm. Gestational exposure of rats to higher concentrations of PCE (900 ppm) were associated with minor behavioral and neurochemical alterations in some offspring, but this may also be a reflection of maternal nutritional deprivation rather than a direct effect of PCE (Nelson et al., 1980 cited in ATSDR, 1990).

Reproductive Toxicity

Limited information is available regarding the reproductive effects of inhaled PCE in animals (ATSDR, 1990). Abnormal sperm were observed in mice exposed to 500 ppm; however, definitive evidence that PCE or its metabolites or impurities reached the germinal tissue and actually damaged DNA is not available (Beliles et al., 1980, cited in ATSDR, 1990). Although the results of a dominant-lethal assay with rats were negative (Beliles et al., 1980, cited in ATSDR, 1990), it should be recognized that this is a relatively insensitive assay that is generally thought to measure gross chromosome damage (EPA, 1985a cited in ATSDR, 1990).

Genotoxicity

Tetrachloroethene itself has not been clearly shown to be a mutagen (ATSDR, 1990). Certain commercial and technical preparations have induced positive responses in the Ames bacterial test, a yeast recombogenic assay, a host-mediated assay, and DNA repair assays, but the responses were weak and required high concentrations of the chemical and no clear dose-response relationships could be established (ATSDR, 1990). The ATSDR considers that there is inadequate information to classify PCE as either nonmutagenic or mutagenic (ATSDR, 1990).

Carcinogenicity

Epidemiological studies of dry-cleaning workers suggest a possible association between chronic PCE exposure and increased cancer risk, but the likelihood of concomitant exposure to petroleum solvents and the effects of other confounding factors and methodological limitations make the results of these studies inconclusive (ATSDR, 1990).

The carcinogenicity of PCE is documented in animals. In chronic inhalation studies, PCE exposure was associated with a higher-than-expected incidence of mononuclear cell leukemia in rats and hepatocellular adenomas and carcinomas in mice (NTP, 1986 cited in ATSDR, 1990). In

chronic oral studies PCE produced hepatocellular carcinomas in mice, but not in rats (NCI, 1977 cited in ATSDR, 1990).

It has been hypothesized that differences in the hepatocarcinogenicity of PCE between rats and mice are due to species differences in peroxisome proliferation resulting from higher trichloroacetic acid blood levels in mice. Trichloroacetic acid results from the biotransformation of PCE and has been shown to induce liver peroxisome proliferation in mice but not in rats (Goldworthy and Popp, 1987 cited in ATSDR, 1990). These findings, together with supporting evidence showing the mutagenicity of PCE epoxide (the highly reactive metabolite of PCE), constitute a "sufficient" level of evidence for the carcinogenicity of PCE in animals using the EPA (1986 cited in ATSDR, 1990) weight-of-evidence criteria. It must be noted, however, that the strains of rats and mice used in these studies have a high spontaneous incidence of tumors and the mechanism of tumor formation in these animals may not be operative in humans. Overall, considering the inconclusive evidence for carcinogenicity in humans, EPA has recommended a Group B2 weight-of-evidence classification.

According to the Region IX EPA, the EPA ECAO has recommended an oral slope factor of 5.2E-02 $(mg/kg/day)^{-1}$ for PCE and an inhalation slope factor of 2.0E-03 $(mg/kg/day)^{-1}$ (EPA Region IX, 1999). Cal EPA recommends an oral slope factor of 2.1E-02 $(mg/kg/day)^{-1}$ and an inhalation slope factor of 5.1E-02 $(mg/kg/day)^{-1}$ (Cal EPA, 1994).

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TOLUENE

(CAS RN 108-88-3)

Toluene is a colorless liquid used as a solvent, raw material, or thinner in chemical, rubber, paint, and drug industries. It is naturally occurring in crude oil and the tolu tree. Toluene is insoluble, but moderately volatile, in water. Toluene is sufficiently volatile that the majority of the toluene in the environment exists in air (ATSDR, 1989).

Absorption/Distribution/Metabolism/Excretion

Toluene is rapidly absorbed following inhalation and less rapidly following ingestion. Absorption of toluene through the skin is less rapid and more limited. Up to 96 percent of an inhaled dose may be retained, of that 20 percent will be excreted unchanged, and 85 percent will be metabolized by the liver.

Because of toluene's lipophilic properties, the highest concentrations of toluene are achieved in the fatty tissues. Immediately after inhalation toluene was found in body fat, bone marrow, spinal nerves, spinal cord, and white matter of the brain; lesser amounts were found in the blood, kidney, and liver (Bergman, 1979 cited in ATSDR, 1989). Very high concentrations of nonvolatile radioactivity, suggesting a toluene conjugate, were found in the kidney and liver.

Toluene is converted to benzyl alcohol by cytochrome P-450 enzymes, rapidly oxidized to benzoic acid by the mixed function oxidases of the liver, and finally conjugated with glycine or hippuric acid prior to excretion by the kidney.

Sixty to 75 percent of an absorbed dose of toluene is excreted by the kidney as hippuric acid conjugate (Ogata et al., 1970 cited in ATSDR, 1989). The majority of a dose is eliminated 12 hours after exposure. The half-life for toluene in the adipose tissue of humans has been estimated at 0.5 to 2.7 days (Carlsson and Ljungquist, 1982 cited in ATSDR, 1989).

Acute Toxicity

The majority of toxicity information on toluene is derived from inhalation exposure studies; there are few or no studies on the effects of oral or dermal exposure to toluene in humans or animals. CNS effects, irritation of the respiratory tract, disturbance of vision, nausea, cardiac effect, and possibly death have been observed in humans and animals following acute inhalation exposures to toluene. The severity of the symptoms increased with the level of exposure (ATSDR, 1989). In monkeys, cognitive function and motor abilities were impaired following acute inhalation exposure to concentrations below that at which ataxia and tremors were observed (Hartman et al., 1984 cited in ATSDR, 1989). Acute oral toxicity of toluene has been studied in rats; very high doses were necessary to achieve death (Kimura et al, 1971 cited in ATSDR, 1989).

Target Organ Toxicity

Information on the toxicity of toluene in humans is derived from epidemiologic studies of solvent abusers or occupationally-exposed workers. Chronic exposure is associated with CNS effects and impaired neuromuscular function; permanent damage has been reported in long-time abusers of toluene (ATSDR, 1989). Toluene has been extensively studied in animals, usually by the inhalation route of exposure. As with humans, the predominate toxic effects of toluene in animals are on the CNS. Oral administration of toluene for 13 weeks or 6 months was associated with minimal toxicity (NTP, 1989; Wolf et al., 1956). Toluene was associated with abnormal skin conditions in humans and was irritating to animal skin following dermal application (EPA, 1983; Wold et al., 1956; Hazleton Laboratories, 1962).

The EPA has established an oral RfD of 2 0E-1 mg/kg/day based on observed increases in liver and kidney weights in a 13-week rat gavage study (NTP, 1989 cited in IRIS, 2000). Rats were exposed to 0, 312, 625, 1,250, 2,500, or 5,000 mg/kg 5 days/week for 13 weeks. The highest dose was lethal to all animals within the first week of administration; some lethality was observed at 2,500 mg/kg. Clinical signs of toxicity, prostration, hypoactivity, ataxia,

piloerection, lacrimation, excessive salivation, and body tremors, were observed in the 2,500 mg/kg dose group. The observed increase in liver and kidney weights was accompanied by histopathologic lesions at the higher doses. The UF of 1000 associated with this RfD is due to the following: inter- and intraspecies extrapolations, subchronic-to-chronic extrapolation, and limited reproductive and developmental toxicity data. Adverse histopathology was also observed in the brains from rats at the two highest doses. In an identical 13-week study in mice, death and clinical signs of toxicity were observed in the two high dose groups; no other adverse findings were reported.

The EPA has established an inhalation RfC of 4.0E-01 mg/m^3 (IRIS) based on observation of neurological effects in humans in an occupational study and degeneration of nasal epithelium in a 2-year rat inhalation study; this yields an inhalation RfD of 1.1E-01 mg/kg/day. Female workers were exposed to toluene emitted from glue in an electronics plant; workers exposed to the toluene-containing glue performed poorly on a battery of 8 neurobehavioral tests (Foo et al., 1990 cited in IRIS, 2000). Although this study was flawed, the results obtained are supported by a number of other human studies. (See preceding section on neurotoxicity.) Dose-related mild to moderate degeneration of olfactory and respiratory epithelium, inflammation of the nasal mucosa, and respiratory metaplasia of the olfactory epithelium were the only toluene-related changes observed in mice and rats following inhalation exposure for 2 years (NTP, 1990 cited in IRIS, 1993). The UF of 300 for this RfC results from a factor of 10 for intraspecies variability, a factor of 10 for LOAEL use, and a factor of 3 for data base deficiences.

Respiratory Toxicity. Irritation of the upper respiratory tract has been observed in humans following chronic (Parmeggiani and Sassi, 1954; Hellquist et al., 1983; Winchester and Madjar, 1986 cited in ATSDR, 1989), but not acute, exposure (von Oettingen et al., 1942 cited in ATSDR, 1989). Variable results have been obtained in rats exposed to toluene for several weeks (von Oettingen et al., 1942; Bruckner and Peterson, 1981; NTP, 1989 cited in ATSDR, 1989). In a 2-year inhalation exposure study, mice and rats developed degenerative changes in olfactory and respiratory epithelium (NTP, 1989 cited in IRIS, 1993). The respiration tract is considered a target organ of toluene toxicity.

Cardiovascular Toxicity. Although cardiac arrhythmia is a cause of death due to solvent abuse, this probably represents an acute pharmacologic effect rather than a direct toxicity. In animal studies, toluene was not directly toxic to the cardiovascular system; histopathological lesions were not observed.

Hematologic Toxicity. A reversible decrease in leukocyte counts was observed in humans and animals exposed to toluene; however, no adverse hematologic effects have been reported (ATSDR, 1989).

Immunotoxicity. Increased susceptibility to respiratory infection was observed in mice exposed to 2.5 to 500 ppm toluene for 5 days; this is probably the result of the cytotoxicity of toluene of alveolar macrophages (Aranyi et al , 1985; Suleiman, 1987 cited in ATSDR, 1989). No reliable data on the immunologic effects of toluene in humans are available.

Hepatotoxicity/Nephrotoxicity. Liver and kidney weights were increased and histopathologic lesions observed in rats exposed to 1250, 2,500, or 5,000 mg/kg 5 days/week for 13 weeks via the drinking water (NTP, 1989 cited in IRIS, 1993)

Neurotoxicity. The primary human health concerns following exposure to toluene are CNS dysfunction and narcosis. This finding is supported by a number of case studies involving solvent abusers and occupationally exposed workers (ATSDR, 1989 and IRIS, 1993). Exposure to low to moderate levels may result in impaired cognitive and neuromuscular function. As the concentration of exposure increases, the symptoms progress to narcosis, e.g., impaired intellectual, psychomotor, and neuromuscular effects. In some cases, the degree of CNS dysfunction is severe enough to result in death. Chronic exposure to high levels have been reported to result in permanent CNS effects, such as ataxia, tremors, atrophy, impaired speech, hearing, and vision, and alterations in EEG activity (Devathasan et al., 1984; King et al., 1981; Suzuki et al., 1983 cited in ATSDR, 1989).

In mice and rats, the extent of CNS damage was correlated with depressed function (Bruckner and Peterson, 1981 cited in ATSDR, 1989). In addition, nystagmus and disturbances in the vestibular and opto-oculomotor systems were reported in rats exposed to toluene (Tham et al., 1982; Larsby et al., 1986 cited in ATSDR, 1989). These data suggest the cerebellum is a target site of toluene. Hippocampal theta wave activity was also disrupted, suggesting an effect on the area of the brain responsible for the integration of information from sensory tissues and organs with responses from visceral and motor control areas (Naaisuno, 1986 cited in ATSDR, 1989). Changes in the levels of brain neurotransmitters have been reported in animals exposed to toluene (Honma et al., 1982; Ikeda et al., 1986; Arito et al., 1985 cited in ATSDR, 1989). Finally, brain weight and total phospholipid content were decreased following inhalation toluene exposure in rats (Kyrklund et al., 1987 cited in ATSDR, 1989), while increased relative brain weight and brain necrosis were observed following oral toluene exposure in mice and rats (NTP, 1989 cited in ATSDR, 1989). Toluene may also exert a toxic effect on the auditory system in animals (Pryor et al., 1984; Johnson et al , 1988; Wood et al., 1983 cited in ATSDR, 1989). The CNS is considered a target organ of toluene toxicity.

Developmental Toxicity

Adverse developmental effects have been reported to occur in humans following in utero exposure. An increased incidence of neural tube defects was observed in children exposed to mixed solvents (including toluene) in utero (Holmberg, 1979). An increase in defects of the urinary tract was associated with children whose mothers were exposed to aromatic solvents, the majority of which were identified as toluene (McDonald et al., 1987 cited in IRIS, 1993). Other developmental effects were reported in 7 children whose mothers were chronic solvent abusers (Goodwin et al., 1988; Hersh et al , 1985; cited in ATSDR, 1989).

In animals, toluene is clearly a developmental toxicant. Skeletal anomalies, retarded skeletal development, low fetal weights, and maternal toxicity were observed following acute exposure to toluene in mice, rabbits, and rats (Courtney, et al , 1986; Ungvary and Tatrai, 1985; Ungvary,

1985; cited in ATSDR, 1989). Toluene was associated with adverse developmental effects when rats were exposed to toluene for 24 but not 6 hours on days 6 through 15 of gestation (API, 1978 cited in ATSDR, 1989). Fetal weight and skeletal retardation were observed in mice that inhaled 1,000 mg/m^3 toluene on days 6 to 15 of gestation (Ungvary and Tatrai, 1985 cited in IRIS, 1993). Embryolethality was reported in mice exposed to 780, 1,300, or 2,600 mg/kg/day on days 6 through 15 of gestation (Nawrot and Staples, 1979 cited in IRIS, 1993). Fetotoxicity, fetal malformations, and maternal toxicity were reported in pregnant rats exposed to 1,000 or 1,500 mg/ m^3 24 hours/day on days 9 through 14 or 1 through 8 or 1 through 21 of gestation (Hudak and Ungvary, 1978 cited in IRIS, 1993). In a two-generation exposure study, growth of the offspring from both generations was inhibited following exposure to 2,000 ppm toluene. Decreased open field activity was observed in mice receiving oral doses of toluene pre- and postnatally; however, this decrease was inversely related to dose administered (Kostas and Hotchin, 1981 cited in ATSDR, 1989). In a study specifically designed to evaluated neurobehavioral effects, no toluene-related adverse findings were observed in the offspring of hamsters or rats following inhalation of 800 mg/m^3 6 hours/day on gestational days 14 through 20 (rats) or 6 through 11 (hamsters) (DaSilva et al., 1990 cited in IRIS, 1993). No developmental effects were observed when pregnant mice were administered oral doses up to 2,350 mg/kg/day (Seidenberg et al., 1986; Smith, 1983 cited in ATSDR, 1989).

Reproductive Toxicity

No reliable data on the reproductive effects of toluene in humans were available.

In animal studies, toluene was without effect on dominant lethal mutations in sperm cells, pre- or post-implantation loss of embryos, or histopathology of the ovaries or testes (API, 1981; CIIT, 1980 cited in ATSDR, 1989). In addition, in a two-generation exposure study, toluene had no effect on survival or reproductive parameters (API, 1985 cited in ATSDR, 1989). Toluene was without effect on the number of implantation sites, liver fetuses, fetal deaths, or fetal body weight in pregnant mice exposed to 200 or 400 ppm in the air on gestational days 7 through 16 (Courtney et al., 1986 cited in IRIS, 1993). No reproductive effects were observed following

administration of a single high oral dose of toluene (Smith, 1983 cited in ATSDR, 1989). Spontaneous abortions and total liter resorptions were reported in mice and hamsters exposed to toluene at levels that caused maternal toxicity, including deaths (Ungvary and Tatrai, 1985 cited in IRIS, 1993).

Genotoxicity

In toluene-exposed workers, the frequency of sister chromatid exchanges was increased (Schmid et al., 1985; Bauchinger et al., 1982 cited in ATSDR, 1989). However, others have reported no increase in the frequencies of chromosome aberrations or sister chromatid exchanges in workers exposed to toluene (Haglund et al., 1980; Maki-Paakkanen et al., 1980; cited in ATSDR, 1989). These human data are limited by the small cohort studied and concurrent exposure to other chemicals. The results of a number of in vitro assays indicate toluene is neither mutagenic or genotoxic (ATSDR, 1989).

Carcinogenicity

The available human data suggest toluene is not carcinogenic; oil refinery workers exposed to toluene and other chemicals had a lower mortality and cancer rate than the general population (Wen et al., 1985 cited in ATSDR, 1989).

The incidence of cancer was not increased in mice or rats exposed to toluene at concentrations up to 1,200 ppm for 2 years (CIIT, 1980; NTP, 1989; cited in ATSDR, 1989). Although the initial study in rats was considered inadequate because the maximum tolerated dose was not used, subsequent studies in mice and rats were considered "well conducted, achieved the maximum tolerated dose, and supports the conclusions of other studies with negative results for carcinogenicity" (ATSDR, 1989). Rats exposed to 0, 600, or 1,200 ppm toluene by inhalation for 6.5 hours/day, 5 days/week exhibited lesions of the nasal cavity, effects on olfactory and respiratory epithelia, and nephropathy, but no evidence of carcinogenicity. However, the EPA has

classified toluene as D, not classified based on no human data and inadequate animal data (IRIS, 1993).

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TOTAL PETROLEUM HYDROCARBONS (TPH)

Petroleum hydrocarbons encompass numerous compounds of varying carbon chain length and arrangement. Within the group of hydrocarbons, individual compounds can be further characterized as belonging to a specific fraction on the basis of boiling point.

The compounds with fewest carbon and highest potential for volatilization include natural gas and some gasoline constituents. Benzene, toluene, ethylbenzene, and xylene (BTEX) are in this fraction. Benzene is a known human carcinogen based on occupational epidemiology. Inhalation is the primary route of exposure and the blood cells the target organ. Leukemia is the resulting disease. Other members of this group have not demonstrated this carcinogenic potential but have been shown to cause adverse effects in the central nervous system and liver.

The middle distillate fraction includes compounds with six or more carbons. Many are used as solvents, thinners, and varnishes. Inhalation of members with this fraction can cause increased respiration, cyanosis and pulmonary edema. The heaviest petroleum fraction includes fuel oils and kerosene. These molecules contain eight or more carbons. These compounds have relatively low to moderate toxicity. Ingestion cause central nervous system depression and gastrointestinal irritation. Dermal exposure can lead to dermatitis.

The polycyclic aromatic hydrocarbons (PAHs) consist of compounds where three or more benzene rings are joined. Benzo(a)pyrene is considered the most toxic of this group because of its association with cancer in animals and human occupational studies. It is postulated that metabolism renders these compounds more toxic. Therefore, the structure of the metabolite influences toxicity.

In the aquatic environment petroleum related compounds may spread as a thin surface layer. Toxicity to birds, mammals, aquatic organisms is incurred through physical contact in which the oils cling to feather, fur epidermis and/or gills. Other hydrocarbons are water soluble and can

form emulsions which may dissolve in the water column or may sink to the bottom sediment where sessile bottom dwelling organisms may be adversely affected.

1,2,4-TRICHLOROBENZENE

(CAS RN 120-82-1)

1,2,4-Trichlorobenzene is a colorless liquid with an aromatic odor. It is mainly used for pesticide production (dicamba, stirofos, and trichlorodinitrobenzene) and as a dye carrier for textiles. 1,2,4-Trichlorobenzene is also used in degreasing agents, septic tank and drain cleaner formulations, wood preservatives, abrasive formulations, and functional fluids (dielectric liquids and transformer oils).

Absorption/Distribution/Metabolism/Excretion

1,2,4-trichlorobenzene is readily absorbed from the gastrointestinal tract following oral administration. When a single 10 mg/kg dose of C-14 labeled compound was orally administered (by gavage) to rats, labeled isomers appeared in blood within 30 minutes post-administration. Blood C-14 levels peaked at approximately 4 hours post-administration, and declined to background within 28 days (HSDB, 1995).

Twenty-four hours after oral administration of C-14 labeled 1,2,4-trichlorobenzene in rats the highest activities were measured in the bladder, kidney, fat, skin, liver, and adrenals (HSDB, 1995). Following administration of 1,2,4-trichlorobenzene in rats at 1 mmol/kg per day for 7 days, the highest levels of 1,2,4-trichlorobenzene were found in fat.

1,2,4-trichlorobenzene undergoes metabolism by phase I oxidation, and phase II glucuronidation or mercapturic acid formation. In monkeys, orally administered 1,2,4-trichlorobenzene is excreted in the urine as a glucuronide conjugate or as the unconjugated trichlorophenol. In rats, 1,2,4-trichlorobenzene metabolites are predominantly excreted in urine as mercapturic acid (60 - 62%), and lesser amounts are excreted as the trichlorothiophenol (28 - 33%) and trichlorophenol (1 - 10%) (HSDB, 1995).

The elimination of radiolabeled (C-14) 1,2,4-trichlorobenzene was studied in rhesus monkeys and rats following oral and intravenous (iv) administration. By 24 hours, monkeys had excreted 40% of the oral dose and 22% of the iv dose in the urine; less than 1% was excreted in feces. For the rat, 84% of the oral dose and 78% of the iv dose were collected in the urine by 24 hours; 11% and 7%, respectively, were collected in the feces (HSDB, 1995).

The halogenated benzenes appear to increase the activity of certain enzyme systems involved in the metabolism of a variety of drugs, pesticides, and other xenobiotics (HSDB, 1995).

Acute Toxicity

Trichlorobenzenes are reported to have less severe toxic effects than dichlorobenzenes. The acute oral LD_{50} for mice is 766 mg/kg, and the LD_{50} for rats is 756 mg/kg (USAF, 1989). The oral lethal dose for a 70-kg human is estimated to be between 50 and 500 mg/kg (USAF, 1989). Following ingestion, nausea, vomiting, and diarrhea may occur (HSDB, 1995).

Target Organ Toxicity.

EPA has assigned an oral RfD of 1.0E-2 mg/kg/day based on increased adrenal weights and vacuolization of zona fasciculata in the cortex. This multigeneration reproductive rat study ended when the F2 generation was 32 days old. Fertility (as indexed by conception rate of dams) of the F0 and F1 generation rats was not affected, but a LOAEL was derived from a significant increase in adrenal gland weights observed in the 400 ppm groups of the F0 and F1 generations. The UF of 1000 associated with the RfD is due to a factor of 10 for extrapolation to humans, 10 for sensitive subpopulations, and 10 for lack of chronic studies (IRIS, 2000).

Hepatic Toxicity. Rats exposed to 1,2,4-trichlorobenzene vapors at levels of 30-100 ppm 7 hours/day, 5 days/week for 30 days had elevated levels of uroporphyrin and coproporphyrin in the urine. No significant effects on body weight, hematology, or pathology were observed at any

dose level. Rats fed 730 mg/kg/day of 1,2,4-trichlorobenzene for 15 days developed hepatic porphyria. (USAF, 1989).

Topical application of 0.5 ml/day of 1,2,4-trichlorobenzene 5 days/week for 3 weeks was lethal to some guinea pigs; livers of these animals showed necrotic foci (USAF, 1989)

In a sub-chronic study, rats were administered 1, 10, 100 or 1,000 ppm of 1,2,4-Trichlorobenzene in corn oil by oral gavage over a period of 90 days. Significant histopathological changes were observed in liver, kidneys, and thyroid only at the highest dose level (1,000 ppm); and the effects were more severe in males than in females HSDB, 1995).

In another sub-chronic study, 1,2,4-Trichlorobenzene was administered in corn oil to male CD rats at doses of 0, 10, 20, or 40 mg/kg/day, and hematological parameters, liver-to-body weight ratios, and induction of enzymes were evaluated. At the highest dose, enzymes were induced and liver-to-body weight ratios increased. These effects, unlike those at lower dose levels, persisted throughout a 30-day recovery period. The 20 mg/kg/day dose was identified as a NOAEL, based on no effects in hematological parameters or liver-to-body weight ratios. This study served as the basis for a chronic RfD developed by the EPA for 1,2,4-trichlorobenzene (USAF, 1989).

Respiratory System Toxicity. A chronic inhalation study was performed in rats, rabbits, and monkeys exposed to 25, 50, and 100 ppm for periods up to 26 weeks. At 4, 13, and 26 weeks, no treatment-related changes were noted in static compliance, carbon monoxide diffusion capacity, distribution of ventilation, transpulmonary pressure, or a battery of lung volume determinations (HSDB, 1995).

Human exposure to levels of 1,2,4-trichlorobenzene in air as low as 3 ppm resulted in respiratory tract irritation. At a level of 2.4 ppm no effects were observed (USAF, 1989). Common reactions of humans to inhalation of 1,2,4-trichlorobenzene include coughing, tachypnea, and wheezing (HSDB, 1995).

Dermal/Ocular Toxicity. Liquid 1,2,4-trichlorobenzene is irritating and causes severe pain on contact with eyes (HSDB, 1995). Human exposure to 3 ppm in air resulted in eye irritation. At a level of 2.4 ppm no effects were observed (USAF, 1989).

Dermal application of technical grade trichlorobenzene in doses of 30, 150, or 450 mg/kg to the backs of rabbits once a day, 5 days/week for 30-31 days caused dermal irritation at the site of application (USAF, 1989). Concentrations of 5%, 25%, or 100% technical-grade 1,2,4-trichlorobenzene were applied topically in 0.2 mL increments to rabbits' ears 3 times a week for 13 weeks. Dermal effects included redness and scaling at the 5% level and severe scaling, encrustation, and desquamation at the 2 upper levels (USAF, 1989). Spongiosis, acanthosis, and parakeratosis were observed in rabbits and guinea pigs, and inflammation of the superficial dermis was observed in rabbits exposed to 1,2,4-trichlorobenzene for three weeks (HSDB, 1995).

Dermal irritation from cutaneous exposure to 1,2,4-trichlorobenzene is probably due to its defatting action. 1,2,4-Trichlorobenzene does not cause chloracne or acneform (HSDB, 1995). Prolonged or repeated contact with liquid chlorinated benzens may result in skin burns (HSDB, 1995).

Developmental Toxicity

Embrotoxic effects of 1,2,4-trichlorobenzene in rats and mice were observed only at concentrations producing maternal toxicity. Teratogenic effects were not observed (USAF, 1989). When 1,2,4-trichlorobenzene was orally administered to rats in doses of 0, 36, 120, 360, or 1200 mg/kg/day on days 9 through 13 of gestation, maternal deaths were observed in the 360 and 1200 mg/kg groups. No increases in teratogenicity or embryolethality were reported, but embryonic development was significantly retarded at 360 mg/kg/day. At the 120 and 360 mg/kg levels, marked maternal liver enzyme induction was observed. At the lowest dosage level there were no observed effects.

In another study, pregnant rats received doses of 150 or 300 mg/kg/day on days 6 through 15 of gestation. There were no signs of maternal toxicity, although increased maternal liver weights and mild thyroid changes were observed. Histology revealed eye lens lesions in fetuses (HSDB, 1995). Oral administration of 360 mg/kg/day to pregnant rats resulted in maternal toxicity and reduced embryonic development (USAF, 1989). Following exposure of rats to 400 ppm 1,2,4-trichlorobenzene in drinking water, enlargement of the adrenal glands was observed in parents and in offspring at 95 days of age (USAF, 1989).

Genotoxicity

Studies utilizing Salmonella typhimurium reported negative results in as many as seven strains, with or without metabolic activation. However, this system is generally insensitive to chlorinated compounds. 1,2,4-trichlorobenzene was positive in an *in vivo* assay for clastogenicity using 8 week old NMRI mice. The mice were injected intraperitoneally with doses of 105, 210, 315, or 420 mg of 1,2,4-trichlorobenzene/kg body weight at 0 and 24 hours and sacrificed at 30 hours. The number of micronucleated cells in femoral bone marrow of treated males showed a positive, statistically significant ($p<0.01$), dose-related response (USAF, 1989).

Carcinogenicity

There are no adequate studies available on the possible carcinogenic effects of 1,2,4-trichlorobenzene (USAF, 1989). EPA has listed 1,2,4-trichlorobenzene as not classifiable as to human carcinogenicity (Group D) based on one chronic, dermal mouse study that was deemed to be inadequate (IRIS, 1995). 1,2,4-Trichlorobenzene in acetone (30 and 60% doses) was applied to the dorsal skin of Slc:ddy mice twice a week for 2 years. Mean survival days were significantly reduced in the males and females that had the 60% dose and in the females that had the 30% dose. Increases of nonneoplastic lesions were found in the lung, liver, kidney, adrenal, spleen, and lymph node of the male high-dose group, and in all the aforementioned organs except the lymph node of the female high-dose group. No single tumor type was increased significantly

over the control EPA noted several limitations in the study, including the following: mice were treated only twice a week, no pharmacokinetic studies were performed, there was a low survival rate, and males were housed individually while females were grouped together (IRIS, 1995). In another study, mice were fed 1,2,4-trichlorobenzene (600 mg/kg diet) for 6 months, and examined for liver tumors. No treatment-related changes in the incidence of hepatomas were observed (USAF, 1989).

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TRICHLOROETHENE

(CAS RN 79-01-6)

Trichloroethene (TCE) is a colorless liquid that is virtually insoluble in water. It is widely used as a metal degreasing agent, in dry-cleaning processes, as a solvent, in organic synthesis, in refrigerants, and in fumigants. In addition, pharmaceutical grade trichloroethene has been used as a general anesthetic in surgical, dental and obstetrical procedures. It is now a ubiquitous environmental pollutant.

Absorption/Distribution/Metabolism/Excretion

As summarized in ATSDR (1989), absorption of TCE following inhalation exposure in humans is characterized by a high initial rate of uptake. Retention of inhaled TCE is independent of inhaled concentration and has been measured at between 37 to 75 percent of the amount inhaled (Nomiyama and Nomiyama, 1971; Soucek and Vlachova, 1960; Bartonicek, 1962 cited in ATSDR, 1989). Although retention is independent of TCE concentration, the absorbed dose in humans has been demonstrated to be directly proportional to inhaled TCE concentration (Astrand and Ovum, 1976 cited in ATSDR, 1989). This proportionality between absorbed dose and inhaled concentration has been observed to break down in animal studies where exposure to higher concentrations of TCE (i.e., 600 ppm) results in metabolic saturation (Stott et al., 1982 cited in ATSDR, 1989)

Absorption of TCE following oral exposure in both humans and animals is rapid and extensive. In animal studies, absorption of TCE from the GI tract has been measured at 91 to 98 percent of TCE (Provt et al., 1985 cited in ATSDR, 1989), and peak TCE blood levels are attained within a matter of hours. Dermal absorption of TCE in both humans and animals is poor, but dermal absorption studies are complicated by the fact that pure liquid TCE can act to defat the skin and thereby enhance its own absorption (Stewart and Dodd, 1964; Sato and Nakajima, 1978; Tsurata, 1978; Jakobson et al., 1982 cited in ATSDR, 1989).

TCE is extensively metabolized (40 to 75 percent of the retained dose) in humans to trichloroethanol, trichloroethanol glucuronide, and trichloroacetic acid (ATSDR, 1989). Minor metabolites include chloral hydrate, monochloroacetic acid, and n-(hydroxyacetyl) aminoethanol. Saturation of metabolism has not been demonstrated in humans up to an exposure concentration of 300 ppm (Nomiyama and Nomiyama, 1977; Ikeda, 1977 cited in ATSDR, 1989). Mathematical models predict, however, that saturation of metabolism is possible at TCE concentrations previously used for anesthesia (i.e., 2,000 ppm) (Feingold and Holaday, 1977 cited in ATSDR, 1989). The major metabolites of TCE in animals are the same as for humans (i.e., trichloroethanol, trichloroethanol glucuronide, and trichloroacetic acid). In addition, animals create a large number of minor TCE metabolites that have not been demonstrated in humans. Although the liver is the primary site of TCE metabolism in animals, there is evidence for extrahepatic metabolism in the kidneys and lungs (Bergman, 1983 cited in ATSDR, 1989). Saturation of metabolism of TCE has been demonstrated in both rats and mice.

In humans, a relatively small amount of absorbed TCE is exhaled through the lungs, while most of the absorbed dose is metabolized and excreted in the urine (ATSDR, 1989). There is a long biological half-life of elimination of TCE from the adipose tissue. Oral studies in rats and mice indicate that the biological half-life of elimination of TCE from the blood is between 1.75 and 2.25 h (D'Souza et al., 1985 cited in ATSDR, 1989).

Acute Toxicity

Acute toxicity data indicate that TCE is relatively nontoxic by the inhalation and oral routes. In mice, LC_{50}'s ranged from 7,480 to 49,000 ppm, whereas in rats the range was 12,500 to 26,300 ppm. Acute LD_{50}'s in dogs, cats, rats, mice, and rabbits ranged from approximately 2,000 to 8,000 mg/kg. Ingestion of 7,000 mg TCE per kg of body weight has been reported to be fatal in humans (ATSDR, 1989).

Target Organ Toxicity

EPA Region IX cites the EPA ECAO as its source for a published oral RfD of 6.0E-03 for TCE (EPA Region IX, 1999).

Neurotoxicity. Inhalation exposure to TCE produces depression (narcosis) and other CNS effects in humans (ATSDR, 1989). The effects (e.g., drowsiness, decreased performance on neurological and psychomotor tests, fatigue, headache, dizziness, ataxia) are well characterized in humans, but exposures associated with the effects are not precisely quantified. The preponderance of information regarding CNS effects in humans (which are summarized in ATSDR, 1989) comes from case studies of unquantified accidental or intentional exposures and from workplace surveys that are limited by typical problems related to exposure quantification (e.g., lack of monitoring data) and control groups. Although many of these reports do not adequately correlate effects with exposure levels, data from the workplace surveys and behavioral performance studies of experimentally exposed humans allow estimation of thresholds for CNS effects.

Grandjean et al. (1955 cited in ATSDR, 1989) studied 50 workers who were exposed to TCE via various industrial cleaning and degreasing operations for an average of 3.75 years. There was higher incidence of neurological disorders in workers in a high-exposure group (85 ppm) compared to that in a low-exposure group (14 or 35 ppm), which suggests that neurological effects in humans may occur at inhalation concentrations of greater than or equal to 85 ppm TCE. Behavioral changes such as reduction in activity were seen in rats at a TCE vapor concentration as low as 100 ppm, which suggest that behavioral alteration is the most sensitive end point for TCE-induced neurological effects in rats (Silverman and Williams, 1975 cited in ATSDR, 1989). An analogy can be drawn between the reduced activity seen in rats after TCE exposure and drowsiness seen in occupationally exposed workers.

Hepatic Toxicity. The liver appears to be a target organ of TCE toxicity in humans. Cases of severe liver damage, including necrosis, from acute occupational exposure to high concentrations

have been reported and are summarized by the EPA (1985b cited in ATSDR, 1989). Based upon this acute human overexposure information and limited animal data, EPA (1985b cited in ATSDR, 1989) has concluded that it is unlikely that chronic exposure to TCE at concentrations found or expected in ambient air would result in liver damage.

Liver enlargement is the most commonly observed hepatic effect seen in TCE exposed animals, indicating that TCE is not as potent a liver toxin as some other chlorinated hydrocarbons. Histological alterations characterized by cellular hypertrophy have been associated with liver enlargement in some studies. Kjellstrand et al. (1983a cited in ATSDR, 1989) reported increased liver weights, enlarged and vacuolated hepatocytes, and increased serum butrylcholinesterase (BuChE) activity in mice exposed to 75 to 300 ppm TCE continuously for 30 days. Increased liver weight and hypertrophy may not be toxic effects since they may be due to the induction of metabolic enzymes, which is generally considered to be an adaptive response rather than an adverse effect. The function of BuChE is not known, but changes in its plasma activity have been noted in the presence of liver disease and may represent early stages of fatty metamorphosis. Nevertheless, the liver effects observed in animals exposed by inhalation to TCE are mild and reversible.

Mice, especially males, appear to be particularly sensitive to the hepatic effects of TCE. Differences in sensitivity among different mouse strains have also been observed (Kjellstrand et al., 1983b cited in ATSDR, 1989). Differences in the hepatic effects of TCE between mice and rats may be attributable to increased metabolism of TCE by mice (Stott et al., 1982 cited in ATSDR, 1989).

Data of Kjellstrand et al. (1983a cited in ATSDR, 1989) show that inhalation exposure using varying time periods and concentrations resulting in exposures approximately equivalent to a 24-h time-weighted average level of 150 ppm produce approximately the same level of hepatic effects in mice. This is consistent with the concept that the toxic effects are due to the formation of metabolites and that the mouse liver has the capability to efficiently metabolize TCE up to very high doses. It should be noted that consumption of alcohol can make the liver susceptible to

injury by TCE and may contribute to or account for the liver effects often associated with TCE per se.

Renal Toxicity. Enlargement is the renal effect most commonly associated with acute or intermediate-duration oral exposure to TCE in rodents (ATSDR, 1989). Kidney enlargement appears to be less pronounced and occurs less consistently than liver enlargement and was associated with altered renal function indices, but not abnormal histology, in some of the intermediate-duration studies.

In contrast to the intermediate-duration studies, chronic inhalation or oral exposures to TCE in mice and rats have produced histological alterations that are characterized by renal tubular alterations and/or toxic nephropathy (ATSDR, 1989). Toxic nephropathy has been observed in orally treated rats and is dissimilar to the chronic nephropathy that is commonly encountered in aging rats (NTP, 1989a cited in ATSDR, 1989).

Respiratory System Toxicity. As summarized in ATSDR (1989) TCE characteristically causes increased respiratory rate (tachypnea) and decreased alveolar ventilatory amplitude when inhaled in anesthetic concentrations, but causes little or no irritation to the respiratory tract (EPA, 1985b; Dobkin and Byles, 1963 cited in ATSDR, 1989). The tachypnea and decreased alveolar ventilatory amplitude are associated with decreased blood oxygen tension and increased carbon dioxide tension.

Other respiratory system effects include reduced lung NADPH cytochrome C reductase activity, vacuolization of bronchiolar epithelial cells in lungs of mice exposed to 10,000 ppm TCE 4 hr/day for 5 days (Lewis et al., 1984 cited in ATSDR, 1989), and reduced pulmonary surfactant secretion in rats exposed to 8,730 ppm 30 min/day for 5 to 15 days (Stewart et al., 1979; Le Mesurier et al., 1980 cited in ATSDR, 1989).

Hematological Toxicity. Continuous exposure to rats to levels of TCE greater than or equal to 50 ppm, or greater than or equal to 398 ppm for 10 days resulted in depression of delta-

aminolevulinate dehydratase (ALA-D) activity in liver, bone marrow cells, and erythrocytes (Fujita et al., 1984 cited in ATSDR, 1989). Related effects included increased ALA-synthetase activity and reduced heme saturation of tryptophan pyrolase in the liver at greater than or equal to 50 ppm, increased urinary excretion of ALA acid at greater than or equal to 398, and coproporphyrin at greater than or equal to 50 ppm. Decreases in ALA-D activity have also been observed in rats (Kolzumi et al., 1984 cited in ATSDR, 1989). This data suggest that TCE exposure may be associated with alteration in heme synthesis.

Cardiotoxicity. The use of TCE as an anesthetic was associated with cardiac arrhythmias, including bradycardia, atrial, and ventricular premature contractions and ventricular extraystoles (EPA, 1985b cited in ATSDR, 1989). Dose-response relationships for effects in humans or animals were not established. Case reports suggested that ingestion of 350 or 500 mL TCE can produce similar effects (Dhuner et al., 1957 cited in ATSDR, 1989).

Animal studies have shown that inhalation of TCE sensitized the heart to epinephrine-induced arrhythmia (EPA, 1985b cited in ATSDR, 1989). This effect was observed in dogs exposed to 5,000 or 10,000 ppm for 10 min (Reinhardt et al., 1973 cited in ATSDR, 1989) and rabbits exposed to 6,000 ppm for 1 h (White and Carlson, 1979, 1981 cited in ATSDR, 1989).

Trichloroethene itself is apparently responsible for the cardiac sensitization, because chemicals that inhibit the metabolism of the parent compound decrease the dose that causes the response, while chemicals that facilitate the metabolism of the parent compound afford protection against the response (White and Carlson, 1979, 1981 cited in ATSDR, 1989). Given the rate of metabolism of TCE and the concentrations required to elicit this effect, sensitization to epinephrine-induced cardiac arrhythmia would not be expected from environmental or normal occupational exposures. Exposures associated with occupational accidents or solvent abuse could be sufficient to cause this effect.

Immunotoxicity. Immunotoxic effects of TCE were evaluated in mice following exposure for 14 days by gavage or 4 to 6 months in the drinking water (Sanders et al., 1982 cited in ATSDR,

1989) Administered doses ranged from 0 to about 793 mg/kg-day. Immunotoxic effects (e.g., delayed type hypersensitivity, decreased bone marrow stem cell colonization) were observed, although the authors noted that they were not remarkable. Nevertheless, this study suggests that TCE may cause immunotoxic effects that are similar to those caused by other chlorinated hydrocarbons (ATSDR, 1989).

Developmental Toxicity

Inhalation studies with rats suggest that TCE is a developmental toxicant. Effects in rats following exposure to 100 ppm TCE for 4 h/day on days 8 to 21 of gestation included completely resorbed litters and indications of delayed fetal development (Healy et al., 1982 cited in ATSDR, 1989). Skeletal anomalies also were observed in fetuses of rats that were exposed to 1,800 ppm for 6 h/day on days 0 to 20 of gestation (Dorfmueller et al., 1979 cited in ATSDR, 1989). In a study in rabbits, some indication was found that TCE induced hydrocephalus in fetuses when the dams were exposed during gestation, but not in litters when dams were exposed during a premating period and gestation (Beliles et al., 1980; Hardin et al., 1981 cited in ATSDR, 1989). Likewise, in the study by Dorfmueller et al. (1979 cited in ATSDR, 1989), no skeletal anomalies were observed in rat fetuses when the dams were exposed before and during gestation, but rather only in litters with gestational exposure. It is possible that some metabolic or physiological tolerance developed during the premating exposure, thus protecting the fetuses from developmental effects.

Genotoxicity

Data from available in vitro and in vivo genotoxicity assays provide suggestive evidence that commercial-grade TCE is a weakly active indirect mutagen (EPA, 1985b cited in ATSDR). Insufficient data are available to allow a conclusion regarding the genotoxic potential of pure TCE. Evaluation of many of the genotoxicity studies of TCE is complicated by the presence of potentially active epoxide stabilizers. The general requirement for metabolic activation, however, argues against the possibility that stabilizers are responsible for the effects and suggest

the involvement of one or more metabolites of TCE (EPA, 1985b cited in ATSDR). Potentially genotoxic metabolites of TCE include trichloroethanol and chloral hydrate (Waskell, 1978; Gu et al., 1981 cited in ATSDR).

Carcinogenicity

Available evidence indicates that TCE is carcinogenic in animals (ATSDR, 1989). Inhalation and/or oral exposure produced liver and lung tumors in mice and kidney adenocarcinomas, testicular Leydig cell tumors, and possibly leukemia in rats. The occurrence of tumors in some of the studies may be influenced by the use of TCE-containing epoxide stabilizers, particularly epichlorohydrin. The available human studies do not allow definite conclusions concerning the carcinogenic potential of TCE in humans.

The available carcinogenicity studies indicate that mice are more susceptible to carcinogenicity than rats. Differences in susceptibility could be due to inherent species differences or to quantitative differences in metabolism or pharmacokinetics (Stott et al., 1982 cited in ATSDR). Another factor that may influence the liver tumor response in mice could be the more pronounced trichloroacetic-acid-mediated peroxisomal proliferation and cell proliferation in mice (Elcombe et al., 1985 cited in ATSDR, 1989). The peroxisomal proliferation may lead to an increase in the reactive oxygen species and DNA damage, which may lead to hepatocellular carcinoma in rodents. Goldworthy et al. (1986 cited in ATSDR, 1989) found that, following treatment with TCE, peroxisomal proliferation was induced in the livers of mice, but not rats, and in the kidneys of both rats and mice. From these results, Goldsworthy et al. (1986) suggested that factors other than peroxisomal proliferation are critical to the carcinogenic response in the rat kidney.

Cal EPA has published an oral slope factor of 1.5E-02 $(mg/kg/day)^{-1}$ and an inhalation slope factor of 1.0E-02 $(mg/kg/day)^{-1}$ for TCE (Cal EPA, 1994). Region IX EPA cites ECAO as the source of an oral slope factor of 1.1E-02 $(mg/kg/day)^{-1}$ and an inhalation slope fact or 6.0E-03 $(mg/kg/day)^{-1}$ (EPA, 1999).

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ZINC

(CAS RN 7440-66-6)

Metallic zinc and various zinc salts are used extensively in the metal plating industry. Zinc is used in galvanizing, electroplating, dry cells, alloying, soldering fluxes, smoke generators, and zinc oxide is used as a pigment for paints and cosmetics. The toxicity of zinc depends on the particular form and salt of zinc and on the route of administration. Zinc is an essential nutrient and is found in a number of metalloenzymes including carbonic anhydrase, carboxypeptidase, alcohol dehydrodgenase, lactic dehydrogenase and alkaline phosphatase (Klaassen et al., 1986).

Zinc salts are used therapeutically as topical astringents, antiseptics, and emetics. Zinc undecylenate is commonly used in athlete's foot preparations and zinc pyridinethione is a common ingredient in antidandruff shampoos. Zinc is often used as a carrier salt for insulin preparations and bactericides.

Acute Effects

Acute exposure to zinc salts via ingestion results in nausea and gastrointestinal irritation. The most common form of acute zinc intoxication is in the inhalation of metal fumes. Zinc fume intoxication results in ocular, mucous membrane, and dermal irritation, malaise, fatigue, headache, blurred vision, and muscle cramping with pulmonary edema at very high concentrations. Upper respiratory tract irritation and sore throat are common symptoms of fume exposures. Acute, lethal exposure results in severe pulmonary edema followed by cyanosis, coma and death (Proctor, 1989).

Chronic Effects

In brass foundry workers, zinc oxide was found to produce zinc fume fevers due to inhalation of fumes during manufacturing processes. Clinical recovery is usually complete in 24 to 48 hours.

Chronic exposure to fumes has not shown adverse effects (NIOSH, 1986). Chronic exposure often results in bronchial pneumonia.

A fine salt of strong mineral acids can be corrosive to the skin and irritating to the gastrointestinal tract. However, the use of zinc oxide in many topical dermatologic preparations has demonstrated a low potential for skin irritation. An occupational dermatitis "Oxidepox" was reported in alloy workers exposed to zinc oxide particulates (ATSDR, 1989). It was concluded that zinc oxide particulates and lack of personal hygiene contributed to the minor eruptions. These were reversible with the institution of good hygiene practices (Clayton, 1981).

Gastrointestinal disturbances with peptic-ulcer-like symptoms have been supported in workers employed for years in brass foundries (Clayton, 1981). Clinically latent liver dysfunction has been reported in workers exposed to high levels of zinc oxide. Evidence of peptic ulcers was felt to be indicative of gastrointestinal tract damage (NIOSH, 1986).

Zinc is a nutritionally essential metal and deficiency results in several health consequences. Excessive exposure to zinc is relatively uncommon and requires very heavy exposure.

Carcinogenic And Other Health Effects

There are no reports of zinc or zinc salts possessing carcinogenic activity. Zinc has been reported to inhibit tumor initiation but the mechanism is unknown. Zinc is cytotoxic to cells in culture and can cause chromosomal abnormalities to mammalian cells in culture

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APPENDIX D

HUMAN HEALTH RISK ASSESSMENT CALCULATIONS

TABLE D-1

CANCER RISK CALCULATIONS FOR MILITARY PERSONNEL
SITE FTIR 32-A - SURFACE SOILS
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration[a] (mg/kg)	Ingestion Dose (mg/kg-day)	Dermal Dose (mg/kg-day)	VOC Inhalation Dose (mg/kg-day)	Dust Inhalation Dose (mg/kg-day)	Cancer Slope Factor $(mg/kg\text{-}d)^{-1}$			Pathway-Specific Cancer Risk				Chemical-Specific Risk
						Oral	Dermal	Inhalation	Ingestion	Dermal	VOC Inhalation	Dust Inhalation	
Metals													
Arsenic	6.3	1.72E-06	9.3E-08	Inc	2.1E-08	1.5E+00	1.5E+00	1.5E+01	2.6E-06	1.4E-07	Inc	3.2E-07	3.0E-06
Cadmium	0.5	1.27E-07	6.9E-09	Inc	1.6E-09	na	na	6.3E+00	0.0E+00	0.0E+00	Inc	9.8E-09	9.8E-09
VOC													
Tetrachloroethylene	0.004	1.03E-09	5.5E-10	3.8E-09	Inc	5.2E-02	5.2E-02	2.0E-03	5.3E-11	2.9E-11	7.6E-12	Inc	9.0E-11
Trichloroethylene	0.003	7.24E-10	3.9E-10	2.3E-09	Inc	1.10E-02	1.10E-02	6.00E-03	8.0E-12	4.3E-12	1.4E-11	Inc	2.6E-11
												ILCR:	3.0E-06

Notes:

[a] Based on the maximum or 95 percent upper confidence limit (95% UCL) on the mean concentration detected at the site.

1) Doses and cancer risks shown only for carcinogenic chemicals with available toxicity values.
2) Absorbed doses were calculated for dermal contact with the medium, and intakes were calculated for ingestion or inhalation of a medium
3) Cancer risks are unitless values which represent the probability of incurring an adverse health effect. They are calculated using the following formula: Cancer Risk = Exposure Dose x Cancer Slope Factor.

ILCR	Incremental lifetime cancer risk
Inc	Incomplete pathway
mg/kg	Milligrams per kilogram
mg/kg-d	Milligrams per kilogram per day
na	Not applicable
VOC	Volatile organic compounds

TABLE D-2

NONCANCER HAZARD CALCULATIONS FOR MILITARY PERSONNEL
SITE FTIR 32-A - SURFACE SOILS
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration[a] (mg/kg)	Ingestion Dose (mg/kg-day)	Dermal Dose (mg/kg-day)	VOC Inhalation Dose (mg/kg-day)	Dust Inhalation Dose (mg/kg-day)	Reference Dose (mg/kg-d) Oral	Dermal	Inhalation	Pathway-Specific Hazard Quotient (HQ) Ingestion	Dermal	VOC Inhalation	Dust Inhalation	Chemical-Specific HQ
Inorganics													
Arsenic	6.3	3.01E-05	1.6E-06	Inc	3.7E-07	3.0E-04	3.0E-04	3.0E-04	1.0E-01	5.4E-03	Inc	1.2E-03	0.11
Barium	125	5.98E-04	3.2E-05	Inc	7.3E-06	7.0E-02	7.0E-02	1.4E-04	8.5E-03	4.6E-04	Inc	5.2E-02	0.061
Cadmium	0.5	2.22E-06	1.2E-07	Inc	2.7E-08	5.0E-04	5.0E-04	5.0E-04	0.0E+00	0.0E+00	Inc	5.4E-05	0.000054
Copper	57	2.74E-04	1.5E-05	Inc	3.3E-06	3.7E-02	3.7E-02	3.7E-02	7.4E-03	4.0E-04	Inc	9.0E-05	0.0079
Manganese	507	2.43E-03	1.3E-04	Inc	3.0E-05	2.4E-02	2.4E-02	1.4E-05	1.0E-01	5.5E-03	Inc	2.1E+00	2.2
Selenium	1.7	8.26E-06	4.5E-07	Inc	1.0E-07	5.0E-03	5.0E-03	5.0E-03	1.7E-03	8.9E-05	Inc	2.0E-05	0.0018
Zinc	328	1.57E-03	8.5E-05	Inc	1.9E-05	3.0E-01	3.0E-01	3.0E-01	5.2E-03	2.8E-04	Inc	6.4E-05	0.0056
VOC													
Tetrachloroethylene	0.004	1.80E-08	9.7E-09	6.6E-08	Inc	1.0E-02	1.0E-02	1.1E-01	1.8E-06	9.7E-07	6.6E-06	Inc	0.000014
Toluene	0.0009	4.31E-09	2.3E-09	7.0E-09	Inc	2.0E-01	2.0E-01	1.1E-01	2.2E-08	1.2E-08	6.3E-08	Inc	0.00000010
Trichloroethylene	0.003	1.27E-08	6.9E-09	4.1E-08	Inc	6.00E-03	6.00E-03	6.00E-03	2.1E-06	1.1E-06	6.8E-06	Inc	0.000010
												Total HI:	2.4

Notes:

[a] Based on the maximum or 95 percent upper confidence limit (95% UCL) on the mean concentration detected at the site.

1) Doses and noncancer hazards shown only for chemicals with available reference doses.
2) Absorbed doses were calculated for dermal contact with the medium, and intakes were calculated for ingestion or inhalation of a medium
3) Noncancer hazards are unitless values which represent the likelihood of incurring an adverse health effect. They are calculated using the following formula: Noncancer Hazard = Exposure Dose / Reference Dose.

HI	Hazard index
HQ	Hazard quotient
Inc	Incomplete pathway
mg/kg	Milligrams per kilogram
mg/kg-d	Milligrams per kilogram per day
na	Not applicable
VOC	Volatile organic compounds

TABLE D-3

CANCER RISK CALCULATIONS FOR A FUTURE INDUSTRIAL WORKER
SITE FTIR-32A - SURFACE SOILS
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration[a] (mg/kg)	Ingestion Dose (mg/kg-d)	Dermal Dose (mg/kg-d)	VOC Inhalation Dose (mg/kg-d)	Dust Inhalation Dose (mg/kg-d)	Cancer Slope Factor (mg/kg-d)$^{-1}$			Pathway-Specific Cancer Risk				Chemical-Specific Risk
						Oral	Dermal	Inhalation	Ingestion	Dermal	VOC Inhalation	Dust Inhalation	
Metals													
Arsenic	6.3	2.20E-06	4.4E-07	Inc	3.3E-10	1.5E+00	1.5E+00	1.5E+01	3.3E-06	6.5E-07	Inc	5.0E-09	4.0E-06
Cadmium	0.5	1.62E-07	1.1E-09	Inc	2.5E-11	na	na	6.3E+00	0.0E+00	0.0E+00	Inc	1.5E-10	1.5E-10
VOC													
Tetrachloroethylene	0.004	1.31E-09	8.6E-10	Inc	2.0E-13	5.2E-02	5.2E-02	2.0E-03	6.8E-11	4.5E-11	Inc	4.0E-16	1.1E-10
Trichloroethylene	0.003	9.24E-10	6.1E-10	Inc	1.4E-13	1.1E-02	1.1E-02	6.0E-03	1.0E-11	6.7E-12	Inc	8.4E-16	1.7E-11
												ILCR:	4.0E-06

Notes:

[a] Based on the maximum or 95 percent upper confidence limit (95% UCL) on the mean concentration detected at the site.

1) Doses and cancer risks shown only for carcinogenic chemicals with available toxicity values.
2) Absorbed doses were calculated for dermal contact with the medium, and intakes were calculated for ingestion or inhalation of a medium
3) Cancer risks are unitless values which represent the probability of incurring an adverse health effect. They are calculated using the following formula: Cancer Risk = Exposure Dose x Cancer Slope Factor.

ILCR	Incremental lifetime cancer risk.
Inc	Incomplete pathway.
mg/kg	Milligrams per kilogram.
mg/kg-d	Milligrams per kilogram per day.
na	Not available.
VOC	Volatile organic compounds

TABLE D-4

NONCANCER HAZARD CALCULATIONS FOR A FUTURE INDUSTRIAL WORKER
SITE FTIR-32A - SURFACE SOILS
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration[a] (mg/kg)	Ingestion Dose (mg/kg-d)	Dermal Dose (mg/kg-d)	VOC Inhalation Dose (mg/kg-d)	Dust Inhalation Dose (mg/kg-d)	Reference Dose (mg/kg-d)			Pathway-Specific Hazard Quotient (HQ)				Chemical-Specific HQ
						Oral	Dermal	Inhalation	Ingestion	Dermal	VOC Inhalation	Dust Inhalation	
Metals													
Arsenic	6.3	6.15E-06	1.2E-06	Inc	9.3E-10	3.0E-04	3.0E-04	3.0E-04	2.1E-02	4.1E-03	Inc	3.1E-06	0.025
Barium	125	1.22E-04	8.1E-06	Inc	1.8E-08	7.0E-02	7.0E-02	1.4E-04	1.7E-03	1.2E-04	Inc	1.3E-04	0.0020
Cadmium	0.5	4.54E-07	3.0E-09	Inc	6.9E-11	5.0E-04	5.0E-04	5.0E-04	9.1E-04	6.0E-06	Inc	1.4E-07	0.00091
Copper	57	5.59E-05	3.7E-06	Inc	8.5E-09	3.7E-02	3.7E-02	3.7E-02	1.5E-03	1.0E-04	Inc	2.3E-07	0.0016
Manganese	507	4.96E-04	3.3E-05	Inc	7.5E-08	2.4E-02	2.4E-02	1.4E-05	2.1E-02	1.4E-03	Inc	5.4E-03	0.027
Selenium	1.7	1.69E-06	1.1E-07	Inc	2.6E-10	5.0E-03	5.0E-03	5.0E-03	3.4E-04	2.2E-05	Inc	5.1E-08	0.00036
Zinc	328	3.21E-04	2.1E-05	Inc	4.9E-08	3.0E-01	3.0E-01	3.0E-01	1.1E-03	7.1E-05	Inc	1.6E-07	0.0011
VOC													
Tetrachloroethylene	0.004	3.67E-09	2.4E-09	Inc	5.6E-13	1.0E-02	1.0E-02	1.1E-01	3.7E-07	2.4E-07	Inc	5.1E-12	0.00000061
Toluene	0.0009	8.81E-10	5.8E-10	Inc	1.3E-13	2.0E-01	2.0E-01	1.1E-01	4.4E-09	2.9E-09	Inc	1.2E-12	0.0000000073
Trichloroethylene	0.003	2.59E-09	1.7E-09	Inc	3.9E-13	6.00E-03	6.00E-03	6.00E-03	4.3E-07	2.8E-07	Inc	6.5E-11	0.00000072
												Total HI:	0.058

Notes:

[a] Based on the maximum or 95 percent upper confidence limit (95% UCL) on the mean concentration detected at the site.

1) Doses and noncancer hazards shown only for noncarcinogenic chemicals with available toxicity values.
2) Absorbed doses were calculated for dermal contact with the medium, and intakes were calculated for ingestion or inhalation of a medium
3) Noncancer hazards are unitless values which represent the likelihood of incurring an adverse health effect. They are calculated using the following formula: Noncancer HI = Exposure Dose/Reference dose.

HI	Hazard index.
Inc	Incomplete pathway.
mg/kg	Milligrams per kilogram.
mg/kd-d	Milligrams per kilogram per day.
VOC	Volatile organic compounds

TABLE D-5

CANCER RISK CALCULATIONS FOR A FUTURE INDUSTRIAL WORKER
SITE FTIR-32A - SUBSURFACE SOILS
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration[a] (mg/kg)	Ingestion Dose (mg/kg-d)	Dermal Dose (mg/kg-d)	VOC Inhalation Dose (mg/kg-d)	Dust Inhalation Dose (mg/kg-d)	Cancer Slope Factor (mg/kg-d)[-1] Oral	Dermal	Inhalation	Pathway-Specific Cancer Risk Ingestion	Dermal	VOC Inhalation	Dust Inhalation	Chemical-Specific Risk
Metals													
Arsenic	9.9	3.44E-06	6.8E-07	Inc	5.2E-10	1.5E+00	1.5E+00	1.5E+01	5.2E-06	1.0E-06	Inc	7.8E-09	6.2E-06
Cadmium	2.5	8.90E-07	5.9E-09	Inc	1.3E-10	na	na	6.3E+00	0.0E+00	0.0E+00	Inc	8.5E-10	8.5E-10
VOC													
Tetrachloroethylene	0.002	6.99E-10	4.6E-10	Inc	1.1E-13	5.20E-02	5.20E-02	2.00E-03	3.6E-11	2.4E-11	Inc	2.1E-16	6.0E-11
Trichloroethylene	0.003	9.55E-10	6.3E-10	Inc	1.4E-13	1.10E-02	1.10E-02	6.00E-03	1.1E-11	6.9E-12	Inc	8.7E-16	1.7E-11
SVOC													
Benzo(a)anthracene	0.014	4.89E-09	4.8E-09	Inc	7.4E-13	7.3E-01	7.3E-01	3.1E-01	3.6E-09	3.5E-09	Inc	2.3E-13	7.1E-09
Benzo(a)pyrene	0.065	2.27E-08	2.2E-08	Inc	3.4E-12	7.3E+00	7.3E+00	3.1E+00	1.7E-07	1.6E-07	Inc	1.1E-11	3.3E-07
Benzo(b)fluoranthene	0.019	6.64E-09	6.6E-09	Inc	1.0E-12	7.3E-01	7.3E-01	3.1E-01	4.8E-09	4.8E-09	Inc	3.1E-13	9.6E-09
bis(2-Ethylhexyl)phthalate	0.2	5.80E-08	3.8E-08	Inc	8.8E-12	1.4E-02	1.4E-02	1.4E-02	8.1E-10	5.4E-10	Inc	1.2E-13	1.3E-09
Chrysene	0.025	8.74E-09	8.6E-09	Inc	1.3E-12	7.3E-03	7.3E-03	3.1E-03	6.4E-11	6.3E-11	Inc	4.1E-15	1.3E-10
Hexachlorobenzene	0.09	3.15E-08	2.1E-08	Inc	4.8E-12	1.6E+00	1.6E+00	1.6E+00	5.0E-08	3.3E-08	Inc	7.6E-12	8.4E-08
Hexachloroethane	0.09	3.15E-08	2.1E-08	Inc	4.8E-12	1.4E-02	1.4E-02	1.4E-02	4.4E-10	2.9E-10	Inc	6.7E-14	7.3E-10
Dioxins/Furans													
TEQ (2,3,7,8)-TCDD	0.00020	7.04E-11	7.0E-11	Inc	1.1E-14	1.5E-05	1.5E-05	1.5E-05	1.1E-15	1.0E-15	Inc	1.6E-19	2.1E-15
												ILCR:	6.6E-06

Notes:

[a] Based on the maximum or 95 percent upper confidence limit (95% UCL) on the mean concentration detected at the site.

1) Doses and cancer risks shown only for carcinogenic chemicals with available toxicity values.
2) Absorbed doses were calculated for dermal contact with the medium, and intakes were calculated for ingestion or inhalation of a medium
3) Cancer risks are unitless values which represent the probability of incurring an adverse health effect. They are calculated using the following formula: Cancer Risk = Exposure Dose x Cancer Slope Factor.

ILCR	Incremental lifetime cancer risk.
Inc	Incomplete pathway.
mg/kg	Milligrams per kilogram.
mg/kg-d	Milligrams per kilogram per day.
na	Not available.
SVOC	Semi-volatile organic compounds
VOC	Volatile organic compounds
TCDD	Tetrachlorodibenzodioxin
TEQ	Toxicity equivalent

TABLE D-6

NONCANCER HAZARD CALCULATIONS FOR A FUTURE INDUSTRIAL WORKER
SITE FTIR-32A - SUBSURFACE SOILS
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration[a] (mg/kg)	Ingestion Dose (mg/kg-d)	Dermal Dose (mg/kg-d)	VOC Inhalation Dose (mg/kg-d)	Dust Inhalation Dose (mg/kg-d)	Reference Dose (mg/kg-d)			Pathway-Specific Hazard Quotient (HQ)				Chemical-Specific HQ
						Oral	Dermal	Inhalation	Ingestion	Dermal	VOC Inhalation	Dust Inhalation	
Metals													
Arsenic	9.9	9.65E-06	1.9E-06	Inc	1.5E-09	3.0E-04	3.0E-04	3.0E-04	3.2E-02	6.4E-03	Inc	4.9E-06	0.039
Barium	167	1.63E-04	1.1E-05	Inc	2.5E-08	7.0E-02	7.0E-02	1.4E-04	2.3E-03	1.5E-04	Inc	1.8E-04	0.0027
Cadmium	2.5	2.49E-06	1.6E-08	Inc	3.8E-10	5.0E-04	5.0E-04	5.0E-04	5.0E-03	3.3E-05	Inc	7.6E-07	0.0050
Copper	1,386	1.36E-03	9.0E-05	Inc	2.1E-07	3.7E-02	3.7E-02	3.7E-02	3.7E-02	2.4E-03	Inc	5.6E-06	0.039
Manganese	471	4.61E-04	3.0E-05	Inc	7.0E-08	2.4E-02	2.4E-02	1.4E-05	1.9E-02	1.3E-03	Inc	5.0E-03	0.025
Mercury	0.09	8.88E-08	5.9E-09	Inc	1.3E-11	3.0E-04	3.0E-04	3.0E-04	3.0E-04	2.0E-05	Inc	4.5E-08	0.00032
Selenium	1.5	1.48E-06	9.8E-08	Inc	2.2E-10	5.0E-03	5.0E-03	5.0E-03	3.0E-04	2.0E-05	Inc	4.5E-08	0.00032
Zinc	3,230	3.16E-03	2.1E-04	Inc	4.8E-07	3.0E-01	3.0E-01	3.0E-01	1.1E-02	7.0E-04	Inc	1.6E-06	0.011
VOC													
1,2-Dichlorobenzene	0.028	2.74E-08	1.8E-08	Inc	4.2E-12	9.0E-02	9.0E-02	5.7E-02	3.0E-07	2.0E-07	Inc	7.3E-11	0.00000051
1,3-Dichlorobenzene	0.062	6.07E-08	4.0E-08	Inc	9.2E-12	9.0E-04	9.0E-04	9.0E-04	6.7E-05	4.4E-05	Inc	1.0E-08	0.00011
Tetrachloroethylene	0.002	1.96E-09	1.3E-09	Inc	3.0E-13	1.0E-02	1.0E-02	1.1E-01	2.0E-07	1.3E-07	Inc	2.7E-12	0.00000032
Trichloroethylene	0.003	2.67E-09	1.8E-09	Inc	4.1E-13	6.0E-03	6.0E-03	6.0E-03	4.5E-07	2.9E-07	Inc	6.8E-11	0.00000074
SVOC													
bis(2-Ethylhexyl)phthalate	0.2	1.62E-07	1.1E-07	Inc	2.5E-11	2.0E-02	2.0E-02	2.0E-02	8.1E-06	5.4E-06	Inc	1.2E-09	0.000013
Di-n-butyl phthalate	0.09	8.99E-08	5.9E-08	Inc	1.4E-11	1.0E-01	1.0E-01	1.0E-01	9.0E-07	5.9E-07	Inc	1.4E-10	0.0000015
Fluoranthene	0.025	2.45E-08	2.4E-08	Inc	3.7E-12	4.0E-02	4.0E-02	4.0E-02	6.1E-07	6.1E-07	Inc	9.3E-11	0.0000012
Hexachlorobenzene	0.09	8.81E-08	5.8E-08	Inc	1.3E-11	8.0E-04	8.0E-04	8.0E-04	1.1E-04	7.3E-05	Inc	1.7E-08	0.00018
Hexachloroethane	0.09	8.81E-08	5.8E-08	Inc	1.3E-11	1.0E-03	1.0E-03	1.0E-03	8.8E-05	5.8E-05	Inc	1.3E-08	0.00015
Naphthalene	0.045	4.40E-08	4.4E-08	Inc	6.7E-12	2.0E-02	2.0E-02	8.6E-04	2.2E-06	2.2E-06	Inc	7.8E-09	0.0000044
Pyrene	0.061	5.97E-08	5.9E-08	Inc	9.0E-12	3.0E-02	3.0E-02	3.0E-02	2.0E-06	2.0E-06	Inc	3.0E-10	0.0000040
1,2,4-Trichlorobenzene	0.042	4.11E-08	2.7E-08	Inc	6.2E-12	1.0E-02	1.0E-02	5.7E-02	4.1E-06	2.7E-06	Inc	1.1E-10	0.0000068
												Total HI:	0.12

Notes:

[a] Based on the maximum or 95 percent upper confidence limit (95% UCL) on the mean concentration detected at the site.

1) Doses and noncancer hazards shown only for noncarcinogenic chemicals with available toxicity values.
2) Absorbed doses were calculated for dermal contact with the medium, and intakes were calculated for ingestion or inhalation of a medium
3) Noncancer hazards are unitless values which represent the likelihood of incurring an adverse health effect. They are calculated using the following formula: Noncancer HI = Exposure Dose/Reference dose.

HQ	Hazard quotient
Inc	Incomplete pathway.
mg/kg	Milligrams per kilogram.
mg/kd-d	Milligrams per kilogram per day.
SVOC	Semi-volatile organic compounds
VOC	Volatile organic compounds

APPENDIX E

BASELINE RISK ASSESSMENT FOR HYPOTHETICAL FUTURE RESIDENTS

E.0 BASELINE RISK ASSESSMENT FOR HYPOTHETICAL FUTURE RESIDENTS

A screening HHRA was conducted as part of the Site Investigation for Sites FTIR-32A and FTIR-39 (Montgomery Watson, 1998). Site FTIR-32A surface and subsurface soils were associated with exceedences of screening risk and hazard criteria for hypothetical future residential (i.e., unrestricted) land uses. Screening cancer risk and noncancer HI estimates for Site FTIR-32A surface soils were 2.6×10^{-5} and 1.1, respectively. Screening cancer risk and HI estimates for Site FTIR-32A subsurface soils were 1.5×10^{-4} and 3.2, respectively. These risk estimates exceed USEPA's acceptable risk range of 1.0×10^{-6} to 1.0×10^{-4}, and HI equal to 1.0 for unrestricted future land uses. However, these screening risk and hazard estimates were based upon a comparison of *maximum* chemical concentrations to USEPA Region IX PRGs, in accordance with DTSC's *Recommended Outline for Using Environmental Protection Agency Region IX Preliminary Remediation Goals in Screening Risk Assessments at Military Facilities* (Cal-EPA, 1994b). Consistent with DTSC guidance (Cal-EPA, 1994b), Site FTIR-32A was further evaluated in a baseline risk assessment and risk estimates were calculated for current/future military personnel and hypothetical future industrial workers (refer to Section 5.0 of this report). However, in comments dated August 31, 2000, DTSC also requested the inclusion of residential risk estimates in the RI Report for Sites FTIR-32A and FTIR-39. Following describes the methods and results of a baseline risk assessment for hypothetical future residential receptors for Site FTIR-32A surface and subsurface soils. Site FTIR-39 was not included in this human health evaluation, because screening cancer risk and noncancer HI estimates for Site FTIR-39 soils were below the screening criteria of 1.0×10^{-6} and 1.0, respectively (Montgomery Watson, 1998).

E.1 METHODS

The general methods used in the baseline HHRA for hypothetical future residents are consistent with the methods described for the evaluation of current military personnel and future industrial workers included in Section 5.0 of this RI Report. Identical COPCs and exposure point concentrations were used in the estimation of risks for hypothetical future residents as were used

in the evaluation of current military personnel and future industrial workers. The COPCs for Site FTIR-32A surface soils and subsurface soils are summarized in Tables 5-2 and 5-3, respectively. The exposure point concentration for each COPC was estimated as either the maximum or 95% UCL on the mean concentration detected in surface or subsurface soils, as described in Section 5.3.5.1. Exposure point concentrations for Site FTIR-32A surface and subsurface soils are summarized in Tables 5-5 and 5-6, respectively.

The algorithms for calculating exposure doses for hypothetical future residents differ from those used for military personnel and future industrial workers in that doses for residents are averaged over the duration of childhood and adult exposures. For hypothetical future residents, the algorithm for calculating the exposure dose due to ingestion of soil is the following:

$$Dose\ (mg/kg\text{-}day) = \frac{((Cs \times IR_{adult} \times EF_{adult} \times ED_{adult} \times UC)/(BW_{adult})) + ((Cs \times IR_{child} \times EF_{child} \times ED_{child} \times UC)/(BW_{child}))}{AT}$$

where:

Cs	=	soil exposure point concentration (mg/kg)
IR_{adult}	=	ingestion rate for adults (mg/day)
IR_{child}	=	ingestion rate children (mg/day)
EF_{adult}	=	exposure frequency for adults (days/year)
EF_{child}	=	exposure frequency for children (days/year)
ED_{adult}	=	exposure duration for adults (years)
ED_{child}	=	exposure duration for children (years)
BW_{adult}	=	body weight for adults (kg)
BW_{child}	=	body weight for children (kg)
UC	=	unit conversion (10^{-6} kg/mg)
AT	=	averaging time (period over which exposure is averaged) (days)

The algorithm for calculating the exposure dose due to dermal contact with soil for hypothetical future residents is the following:

$$\text{Dose (mg/kg-day)} = \frac{((Cs \times AF_{adult} \times SA_{adult} \times ABS \times EF_{adult} \times ED_{adult} \times UC)/(BW_{adult})) + ((Cs \times AF_{child} \times SA_{child} \times ABS \times EF_{child} \times ED_{child} \times UC)/(BW_{child}))}{AT}$$

where:

Cs	=	soil exposure point concentration (mg/kg)
AF_{adult}	=	dermal adherence factor for adults (mg/cm^2)
AF_{child}	=	dermal adherence factor children (mg/cm^2)
SA_{adult}	=	dermal surface area for adults (cm^2/event)
SA_{child}	=	dermal surface area for children (cm^2/event)
EF_{adult}	=	exposure frequency for adults (days/year)
EF_{child}	=	exposure frequency for children (days/year)
ED_{adult}	=	exposure duration for adults (years)
ED_{child}	=	exposure duration for children (years)
BW_{adult}	=	body weight for adults (kg)
BW_{child}	=	body weight for children (kg)
ABS	=	fraction of chemical absorbed (unitless)
UC	=	unit conversion (10^{-6} kg/mg)
AT	=	averaging time (period over which exposure is averaged) (days)

The algorithm for calculating exposure due to inhalation of particulates from soil for hypothetical future residents is the following:

$$\text{Dose (mg/kg-day)} = \frac{((Cs \times (1/PEF) \times InhR_{adult} \times EF_{adult} \times ED_{adult})/(BW_{adult})) + ((Cs \times (1/PEF) \times InhR_{child} \times EF_{child} \times ED_{child})/(BW_{child}))}{AT}$$

where:

Cs	=	soil exposure point concentration (mg/kg)
PEF	=	particulate emission factor (m^3/kg)
$InhR_{adult}$	=	dermal adherence factor for adults (m^3/day)
$InhR_{child}$	=	dermal adherence factor children (m^3/day)
EF_{adult}	=	exposure frequency for adults (days/year)
EF_{child}	=	exposure frequency for children (days/year)
ED_{adult}	=	exposure duration for adults (years)
ED_{child}	=	exposure duration for children (years)
BW_{adult}	=	body weight for adults (kg)
BW_{child}	=	body weight for children (kg)
AT	=	averaging time (period over which exposure is averaged) (days)

The algorithm for calculating exposure due to inhalation of VOCs from soil for hypothetical future residents is the following:

$$Dose\ (mg/kg\text{-}day) = \frac{((Cs \times (1/VF) \times InhR_{adult} \times EF_{adult} \times ED_{adult})/(BW_{adult})) + ((Cs \times (1/VF) \times InhR_{child} \times EF_{child} \times ED_{child})/(BW_{child}))}{AT}$$

where:

Cs	=	soil exposure point concentration (mg/kg)
VF	=	volatilization factor (m^3/kg)
$InhR_{adult}$	=	dermal adherence factor for adults (m^3/day)
$InhR_{child}$	=	dermal adherence factor children (m^3/day)
EF_{adult}	=	exposure frequency for adults (days/year)
EF_{child}	=	exposure frequency for children (days/year)
ED_{adult}	=	exposure duration for adults (years)
ED_{child}	=	exposure duration for children (years)

BW_{adult} = body weight for adults (kg)

BW_{child} = body weight for children (kg)

AT = averaging time (period over which exposure is averaged) (days)

The parameters and assumptions used in modeling exposure doses for hypothetical future residents are summarized in Table E-1. The toxicity values that were used in the characterization of risks for hypothetical future residents are the same as those that were used in the baseline risk evaluation for current military personnel and future industrial workers. These toxicity values were summarized in Table 5-9.

Baseline human health risks for hypothetical future residents were evaluated separately for carcinogenic effects and noncarcinogenic effects. Cancer risks were calculated as the product of the exposure dose and the carcinogenic toxicity value, or CSF (USEPA, 1989), as follows:

ILCR (unitless) = CSF X Dose

where:

CSF = Cancer slope factor (mg/kg-day)-1

Dose = Exposure dose for carcinogens (mg/kg-day)

Cancer risks from multiple COPCs were assumed to be additive, and were summed to estimate a total cumulative ILCR for all carcinogenic site contaminants. Noncancer hazards for hypothetical future residents were calculated as the ratio of the exposure dose to the noncarcinogenic toxicity value, or RfD (USEPA, 1989), as follows:

$$HQ\ (unitless) = \frac{Dose}{RfD}$$

where:

Dose = Exposure dose for noncarcinogens (mg/kg-day)

RfD = Reference dose (mg/kg-day)

The individual HQs for site COPCs were summed to produce total cumulative HI values for FTIR-32A surface and subsurface soils.

E.2 RESULTS

The results of the baseline risk assessment performed for hypothetical future residents exposed to Site FTIR-32A surface and subsurface soils are described in this section. Detailed carcinogenic risk and noncarcinogenic hazard calculations for hypothetical future residents are presented in Tables E-2 through E-5.

The total cumulative cancer risk and noncancer hazard (HI) estimates for hypothetical future residents exposed to surface soils were 1.7×10^{-5} and 1.0, respectively (Tables E-2 and E-3). These cancer risk and HI estimates are within USEPA's generally acceptable cancer risk range of 1.0×10^{-6} to 1.0×10^{-4}, and HI less than or equal to 1.0 (USEPA, 1991a).

The total cumulative cancer risk and noncancer HI values for hypothetical future residents exposed to subsurface soils were 2.8×10^{-5} and 0.73, respectively (Tables E-4 and E-5). These cancer risk and HI estimates are within USEPA's generally acceptable cancer risk range of 1.0×10^{-6} to 1.0×10^{-4}, and HI less than or equal to 1.0 (USEPA, 1991a).

The baseline risk assessment results for all receptors, including hypothetical future residents, are summarized in Section 7.0 and Table 7-1 of this RI Report, and recommendations regarding FTIR-32A are presented.

TABLE E-1

EXPOSURE PARAMETERS AND ASSUMPTIONS FOR HYPOTHETICAL FUTURE RESIDENTS
FORT IRWIN, CALIFORNIA

Exposure Parameter	Units	Exposure Assumption		Source
		Adult	Child	
Soil Concentration - C_s	mg/kg	Chemical-specific	Chemical-specific	Not applicable
Body Weight - BW	kg	70	15	USEPA, 1997
Soil Ingestion Rate - IR	mg/day	100	200	USEPA, 1997
Inhalation Rate - InhR	m^3/day	20	10	USEPA, 1997
Exposure Frequency - EF	day/yr	350	350	USEPA, 1997
Exposure Duration - ED	yr	24	6	USEPA, 1997
Dermal Surface Area - SA	cm^2/event	5.700	2,800	USEPA, 1999a
Skin Adherence Factor - AF	mg/cm^2	0 07	0 2	USEPA, 1999a
Averaging Time - AT	days			
Carcinogens		25,550	25,550	USEPA, 1989
Noncarcinogens		8.760	2,190	USEPA, 1989
Particulate Emission Factor - PEF	m^3/kg	1 6E+07	1 6E+07	Parsons, 1995
Volatilization Factor - VF	m^3/kg			
1,2-Dichlorobenzene		1 2E+04	1 2E+04	USEPA, 1999b
1,3-Dichlorobenzene		1 2E+04	1 2E+04	USEPA. 1999b
Tetrachloroethylene		3 2E+03	3 2E+03	USEPA, 1999b
Trichloroethylene		2 6E+03	2 6E+03	USEPA, 1999b
Toluene		3 6E+03	3 6E+03	USEPA, 1999b

Sources:
Exposure Factors Handbook (USEPA, 1997)
Project Workplan for the Site Inspection and Remedial Investigation of 31 Sites at NTC Fort Irwin (Parsons, 1995)
RAGS, Supplemental Guidance-Dermal Risk Assessment (USEPA, 1999a).
Region 9 Preliminary Remediation Goals (PRGs) 1999 (USEPA, 1999b).
Risk Assessment Guidance for Superfund (RAGS), Volume 1: Human Health Evaluation Manual (Part A) (USEPA, 1989).

cm^2/event-Square centimeters per event
day/yr-Days per year
kg-Kilogram
m^3/day-Cubic meters per day
m^3/kg-Cubic meters per kilogram
mg/cm^2-Milligrams per square centimeter
mg/day-Milligrams per day
mg/kg-Milligrams per kilogram
yr-Year

TABLE E-2

CANCER RISK CALCULATIONS FOR A HYPOTHETICAL FUTURE RESIDENT
SITE FTIR-32A - SURFACE SOILS
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration[a] (mg/kg)	Ingestion Dose (mg/kg-d)	Dermal Dose (mg/kg-d)	VOC Inhalation Dose (mg/kg-d)	Dust Inhalation Dose (mg/kg-d)	Cancer Slope Factor (mg/kg-d)$^{-1}$			Pathway-Specific Cancer Risk				Chemical-Specific Risk
						Oral	Dermal	Inhalation	Ingestion	Dermal	VOC Inhalation	Dust Inhalation	
Metals													
Arsenic	6.3	9.85E-06	9.3E-07	Inc	5.8E-08	1.5E+00	1.5E+00	1.5E+01	1.5E-05	1.4E-06	Inc	8.8E-07	1.7E-05
Cadmium	0.5	7.26E-07	2.3E-09	Inc	4.3E-09	na	na	6.3E+00	0.0E+00	0.0E+00	Inc	2.7E-08	2.7E-08
VOC													
Tetrachloroethylene	0.004	5.87E-09	7.1E-10	1.7E-07	3.5E-11	5.2E-02	5.2E-02	2.0E-03	3.1E-10	3.7E-11	3.5E-10	7.0E-14	6.9E-10
Trichloroethylene	0.003	4.14E-09	5.0E-10	1.4E-07	2.5E-11	1.1E-02	1.1E-02	6.0E-03	4.6E-11	5.5E-12	8.5E-10	1.5E-13	9.0E-10
												ILCR:	1.7E-05

Notes:

[a] Based on the maximum or 95 percent upper confidence limit (95% UCL) on the mean concentration detected at the site.

1) Doses and cancer risks shown only for carcinogenic chemicals with available toxicity values.
2) Absorbed doses were calculated for dermal contact with the medium, and intakes were calculated for ingestion or inhalation of a medium
3) Cancer risks are unitless values which represent the probability of incurring an adverse health effect. They are calculated using the following formula: Cancer Risk = Exposure Dose x Cancer Slope Factor.

ILCR	Incremental lifetime cancer risk.
Inc	Incomplete pathway.
mg/kg	Milligrams per kilogram.
mg/kg-d	Milligrams per kilogram per day.
na	Not available.
VOC	Volatile organic compounds

TABLE E-3

NONCANCER HAZARD CALCULATIONS FOR A HYPOTHETICAL FUTURE RESIDENT
SITE FTIR-32A - SURFACE SOILS
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration[a] (mg/kg)	Ingestion Dose (mg/kg-d)	Dermal Dose (mg/kg-d)	VOC Inhalation Dose (mg/kg-d)	Dust Inhalation Dose (mg/kg-d)	Reference Dose (mg/kg-d)			Pathway-Specific Hazard Quotient (HQ)				Chemical-Specific HQ
						Oral	Dermal	Inhalation	Ingestion	Dermal	VOC Inhalation	Dust Inhalation	
Metals													
Arsenic	6.3	2.30E-05	2.2E-06	Inc	1.4E-07	3.0E-04	3.0E-04	3.0E-04	7.7E-02	7.3E-03	Inc	4.6E-04	0.084
Barium	125	4.56E-04	3.2E-05	Inc	2.7E-06	7.0E-02	7.0E-02	1.4E-04	6.5E-03	4.6E-04	Inc	1.9E-02	0.026
Cadmium	0.5	1.69E-06	1.0E-07	Inc	1.0E-08	5.0E-04	5.0E-04	5.0E-04	3.4E-03	2.0E-04	Inc	2.0E-05	0.0036
Copper	57	2.09E-04	1.5E-05	Inc	1.2E-06	3.7E-02	3.7E-02	3.7E-02	5.6E-03	4.0E-04	Inc	3.4E-05	0.0061
Manganese	507	1.85E-03	1.3E-04	Inc	1.1E-05	2.4E-02	2.4E-02	1.4E-05	7.7E-02	5.5E-03	Inc	7.9E-01	0.87
Selenium	1.7	6.30E-06	4.5E-07	Inc	3.7E-08	5.0E-03	5.0E-03	5.0E-03	1.3E-03	8.9E-05	Inc	7.5E-06	0.0014
Zinc	328	1.20E-03	8.5E-05	Inc	7.1E-06	3.0E-01	3.0E-01	3.0E-01	4.0E-03	2.8E-04	Inc	2.4E-05	0.0043
VOC													
Tetrachloroethylene	0.004	1.37E-08	2.4E-09	1.7E-07	8.1E-11	1.0E-02	1.0E-02	1.1E-01	1.4E-06	2.4E-07	Inc	7.4E-10	0.0000016
Toluene	0.0009	3.29E-09	5.9E-10	4.2E-08	2.0E-11	2.0E-01	2.0E-01	1.1E-01	1.6E-08	2.9E-09	Inc	1.8E-10	0.000000020
Trichloroethylene	0.003	9.66E-09	1.7E-09	1.2E-07	5.7E-11	6.00E-03	6.00E-03	6.00E-03	1.6E-06	2.9E-07	Inc	9.6E-09	0.0000019
												Total HI:	1.0

Notes:

[a] Based on the maximum or 95 percent upper confidence limit (95% UCL) on the mean concentration detected at the site.

1) Doses and noncancer hazards shown only for noncarcinogenic chemicals with available toxicity values.
2) Absorbed doses were calculated for dermal contact with the medium, and intakes were calculated for ingestion or inhalation of a medium
3) Noncancer hazards are unitless values which represent the likelihood of incurring an adverse health effect. They are calculated using the following formula: Noncancer HI = Exposure Dose/Reference dose.

HI	Hazard index.
Inc	Incomplete pathway.
mg/kg	Milligrams per kilogram.
mg/kd-d	Milligrams per kilogram per day.
VOC	Volatile organic compounds

TABLE E-4

CANCER RISK CALCULATIONS FOR A HYPOTHETICAL FUTURE RESIDENT
SITE FTIR-32A - SUBSURFACE SOILS
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration[a] (mg/kg)	Ingestion Dose (mg/kg-d)	Dermal Dose (mg/kg-d)	VOC Inhalation Dose (mg/kg-d)	Dust Inhalation Dose (mg/kg-d)	Cancer Slope Factor (mg/kg-d)[-1]			Pathway-Specific Cancer Risk				Chemical-Specific Risk
						Oral	Dermal	Inhalation	Ingestion	Dermal	VOC Inhalation	Dust Inhalation	
Metals													
Arsenic	9.9	1.54E-05	1.5E-06	Inc	9.2E-08	1.5E+00	1.5E+00	1.5E+01	2.3E-05	2.2E-06	Inc	1.4E-06	2.7E-05
Cadmium	2.5	3.99E-06	1.3E-08	Inc	2.4E-08	na	na	6.3E+00	0.0E+00	0.0E+00	Inc	1.5E-07	1.5E-07
VOC													
Tetrachloroethylene	0.002	3.13E-09	9.9E-10	9.3E-08	1.9E-11	5.20E-02	5.20E-02	2.00E-03	1.6E-10	5.1E-11	Inc	3.7E-14	2.1E-10
Trichloroethylene	0.003	4.28E-09	1.4E-09	1.5E-07	2.5E-11	1.10E-02	1.10E-02	6.00E-03	4.7E-11	1.5E-11	Inc	1.5E-13	6.2E-11
SVOC													
Benzo(a)anthracene	0.014	2.19E-08	1.0E-08	Inc	1.3E-10	7.3E-01	7.3E-01	3.1E-01	1.6E-08	7.6E-09	Inc	4.0E-11	2.4E-08
Benzo(a)pyrene	0.065	1.02E-07	4.8E-08	Inc	6.0E-10	7.3E+00	7.3E+00	3.1E+00	7.4E-07	3.5E-07	Inc	1.9E-09	1.1E-06
Benzo(b)fluoranthene	0.019	2.97E-08	1.4E-08	Inc	1.8E-10	7.3E-01	7.3E-01	3.1E-01	2.2E-08	1.0E-08	Inc	5.5E-11	3.2E-08
bis(2-Ethylhexyl)phthalate	0.2	2.60E-07	8.2E-08	Inc	1.5E-09	1.4E-02	1.4E-02	1.4E-02	3.6E-09	1.1E-09	Inc	2.2E-11	4.8E-09
Chrysene	0.025	3.91E-08	1.9E-08	Inc	2.3E-10	7.3E-03	7.3E-03	3.1E-03	2.9E-10	1.4E-10	Inc	7.2E-13	4.2E-10
Hexachlorobenzene	0.09	1.41E-07	4.4E-08	Inc	8.4E-10	1.6E+00	1.6E+00	1.6E+00	2.3E-07	7.1E-08	Inc	1.3E-09	3.0E-07
Hexachloroethane	0.09	1.41E-07	4.4E-08	Inc	8.4E-10	1.4E-02	1.4E-02	1.4E-02	2.0E-09	6.2E-10	Inc	1.2E-11	2.6E-09
Dioxins/Furans													
TEQ (2,3,7,8)-TCDD	0.00020	3.16E-10	1.0E-10	Inc	1.9E-12	1.5E-05	1.5E-05	1.5E-05	4.7E-15	1.5E-15	Inc	2.8E-17	6.3E-15
												ILCR:	2.8E-05

Notes:

[a] Based on the maximum or 95 percent upper confidence limit (95% UCL) on the mean concentration detected at the site.

1) Doses and cancer risks shown only for carcinogenic chemicals with available toxicity values.
2) Absorbed doses were calculated for dermal contact with the medium, and intakes were calculated for ingestion or inhalation of a medium
3) Cancer risks are unitless values which represent the probability of incurring an adverse health effect. They are calculated using the following formula: Cancer Risk = Exposure Dose x Cancer Slope Factor.

ILCR	Incremental lifetime cancer risk.
Inc	Incomplete pathway.
mg/kg	Milligrams per kilogram.
mg/kg-d	Milligrams per kilogram per day.
na	Not available.
SVOC	Semi-volatile organic compounds
VOC	Volatile organic compounds
TCDD	Tetrachlorodibenzodioxin
TEQ	Toxicity equivalent

TABLE E-5

NONCANCER HAZARD CALCULATIONS FOR A HYPOTHETICAL FUTURE RESIDENT
SITE FTIR-32A - SUBSURFACE SOILS
FORT IRWIN, CALIFORNIA

Constituent	Soil Concentration[a] (mg/kg)	Ingestion Dose (mg/kg-d)	Dermal Dose (mg/kg-d)	VOC Inhalation Dose (mg/kg-d)	Dust Inhalation Dose (mg/kg-d)	Reference Dose (mg/kg-d)			Pathway-Specific Hazard Quotient (HQ)				Chemical-Specific HQ
						Oral	Dermal	Inhalation	Ingestion	Dermal	VOC Inhalation	Dust Inhalation	
Metals													
Arsenic	9.9	3.62E-05	1.5E-06	Inc	9.2E-08	3.0E-04	3.0E-04	3.0E-04	1.2E-01	4.9E-03	Inc	3.1E-04	0.126
Barium	167	6.09E-04	8.2E-06	Inc	1.5E-06	7.0E-02	7.0E-02	1.4E-04	8.7E-03	1.2E-04	Inc	1.1E-02	0.0199
Cadmium	2.5	9.30E-06	1.3E-08	Inc	2.4E-08	5.0E-04	5.0E-04	5.0E-04	1.9E-02	2.5E-05	Inc	4.7E-05	0.0187
Copper	1,386	5.06E-03	6.9E-05	Inc	1.3E-05	3.7E-02	3.7E-02	3.7E-02	1.4E-01	1.9E-03	Inc	3.5E-04	0.139
Manganese	471	1.72E-03	2.3E-05	Inc	4.4E-06	2.4E-02	2.4E-02	1.4E-05	7.2E-02	9.7E-04	Inc	3.1E-01	0.386
Mercury	0.09	3.31E-07	4.5E-09	Inc	8.4E-10	3.0E-04	3.0E-04	3.0E-04	1.1E-03	1.5E-05	Inc	2.8E-06	0.00112
Selenium	1.5	5.52E-06	7.5E-08	Inc	1.4E-08	5.0E-03	5.0E-03	5.0E-03	1.1E-03	1.5E-05	Inc	2.8E-06	0.00112
Zinc	3,230	1.18E-02	1.6E-04	Inc	3.0E-05	3.0E-01	3.0E-01	3.0E-01	3.9E-02	5.3E-04	Inc	1.0E-04	0.040
VOC													
1,2-Dichlorobenzene	0.028	1.02E-07	1.4E-08	3.5E-07	2.6E-10	9.0E-02	9.0E-02	5.7E-02	1.1E-06	1.5E-07	Inc	4.6E-09	0.00000129
1,3-Dichlorobenzene	0.062	2.26E-07	3.1E-08	7.7E-07	5.8E-10	9.0E-04	9.0E-04	9.0E-04	2.5E-04	3.4E-05	Inc	6.4E-07	0.00029
Tetrachloroethylene	0.002	7.31E-09	9.9E-10	9.3E-08	1.9E-11	1.0E-02	1.0E-02	1.1E-01	7.3E-07	9.9E-08	Inc	1.7E-10	0.00000083
Trichloroethylene	0.003	9.98E-09	1.4E-09	1.6E-07	2.5E-11	6.0E-03	6.0E-03	6.0E-03	1.7E-06	2.3E-07	Inc	4.2E-09	0.00000189
SVOC													
bis(2-Ethylhexyl)phthalate	0.2	6.06E-07	8.2E-08	Inc	1.5E-09	2.0E-02	2.0E-02	2.0E-02	3.0E-05	4.1E-06	Inc	7.7E-08	0.000034
Di-n-butyl phthalate	0.09	3.36E-07	4.5E-08	Inc	8.5E-10	1.0E-01	1.0E-01	1.0E-01	3.4E-06	4.5E-07	Inc	8.5E-09	0.0000038
Fluoranthene	0.025	9.13E-08	1.9E-08	Inc	2.3E-10	4.0E-02	4.0E-02	4.0E-02	2.3E-06	4.6E-07	Inc	5.8E-09	0.0000028
Hexachlorobenzene	0.09	3.29E-07	4.4E-08	Inc	8.4E-10	8.0E-04	8.0E-04	8.0E-04	4.1E-04	5.6E-05	Inc	1.0E-06	0.00047
Hexachloroethane	0.09	3.29E-07	4.4E-08	Inc	8.4E-10	1.0E-03	1.0E-03	1.0E-03	3.3E-04	4.4E-05	Inc	8.4E-07	0.00037
Naphthalene	0.045	1.64E-07	3.3E-08	Inc	4.2E-10	2.0E-02	2.0E-02	8.6E-04	8.2E-06	1.7E-06	Inc	4.9E-07	0.0000104
Pyrene	0.061	2.23E-07	4.5E-08	Inc	5.7E-10	3.0E-02	3.0E-02	3.0E-02	7.4E-06	1.5E-06	Inc	1.9E-08	0.0000090
1,2,4-Trichlorobenzene	0.042	1.53E-07	2.1E-08	Inc	3.9E-10	1.0E-02	1.0E-02	5.7E-02	1.5E-05	2.1E-06	Inc	6.8E-09	0.0000174
												Total HI:	0.73

Notes:

[a] Based on the maximum or 95 percent upper confidence limit (95% UCL) on the mean concentration detected at the site.

1) Doses and noncancer hazards shown only for noncarcinogenic chemicals with available toxicity values.
2) Absorbed doses were calculated for dermal contact with the medium, and intakes were calculated for ingestion or inhalation of a medium
3) Noncancer hazards are unitless values which represent the likelihood of incurring an adverse health effect. They are calculated using the following formula: Noncancer HI = Exposure Dose/Reference dose.

HQ	Hazard quotient
Inc	Incomplete pathway.
mg/kg	Milligrams per kilogram.
mg/kd-d	Milligrams per kilogram per day.
SVOC	Semi-volatile organic compounds
VOC	Volatile organic compounds

APPENDIX F

RESPONSES TO COMMENTS

REVIEW COMMENTS ON:
DRAFT REMEDIAL INVESTIGATION REPORT
FOR SITES FTIR-32A AND FTIR-39
NATIONAL TRAINING CENTER, FORT IRWIN, CA

From: Department of Toxic Substances Control (DTSC)

Comments from Omoruyi Patrick, DTSC, Dated 31 August 2000

SPECIFIC COMMENTS

Comment No. 1: *Include the human health risk assessment results for the future resident scenario in Section 5.0 and Table 5-10 for Sites FTIR-32A and FTIR-39 to show the estimated human health risk levels, respectively.*

MW Response: Please note that the baseline human health risk assessment is intended to be a more realistic evaluation of risks than the screening risk assessment, and is based on realistic exposure assumptions and anticipated future land uses (USEPA, 1989; Cal-EPA, 1994). Consistent with USEPA (1989) and Cal-EPA (1994) guidance, the baseline risk assessment that was presented in Section 5.0 of the Remedial Investigation Report for Sites FTIR-32A and FTIR-39 evaluated risks for current military personnel and future industrial workers.

The Army has included risk and hazard estimates for a hypothetical future residential scenario as the DTSC requested, but has presented these risks in Appendix E so as not to alter the intent or conclusions of the baseline risk assessment that is presented in Section 5.0. Additionally, risks for the hypothetical future residential scenario were calculated using the 95 percent upper confidence level on the mean (95% UCL) concentration, rather than the maximum site concentrations that were used in the Site Investigation Report for Sites FTIR-32A, FTIR-38, FTIR-39, and FTIR-40 (Montgomery Watson, 1998). Risks for hypothetical future residential receptors are summarized in Section 7.0 of the revised RI Report, along with the results and conclusions of the baseline risk assessment.

REVIEW COMMENTS ON:
DRAFT REMEDIAL INVESTIGATION REPORT
FOR SITES FTIR-32A AND FTIR-39
NATIONAL TRAINING CENTER, FORT IRWIN, CA

From: Department of Toxic Substances Control (DTSC)

Comments from John Christopher, DTSC, Dated 7 December 2000

GENERAL COMMENTS

Comment No. 1: *The document is clear and well written. In the human health risk assessment, we feel potential risks due to inhalation for the future worker are underestimated. We disagree with the Army's conclusion that risks and hazards due to metals are approximately the same as site background. We agree with the Army's conclusion that no habitat exists for the Mojave ground squirrel at either site.*

MW Response: Cancer risks and noncancer hazards have been re-calculated for the future industrial worker assuming a particulate emission factor (PEF) of 1.6E+07 (refer to the Army's response to Specific Comment No. 2). The statement that risks and hazards due to metals are approximately the same as site background have been revised in the Final RI Report, as indicated in the Army's response to Specific Comment No. 3, below.

Comment No. 2: *Risk and hazard via inhalation should be re-calculated for the future worker, using a higher value for dust inhalation than was used in the current document. Except for this, we concur with the Army's estimates of risk and hazard for both human and non-human species.*

MW Response: Revised cancer risk and noncancer hazard estimates for the future industrial worker, assuming a particulate emission factor (PEF) of 1.6E+07, have been included in the Final RI Report, as indicated in the Army's response to Specific Comment No. 2, below.

SPECIFIC COMMENTS

Comment No 1. *Tables 5-5 and 5-6. Please add units of concentration to these tables. Also please correct the location of Fort Irwin (i e , not Lompoc, CA).*

MW Response: These tables have been revised.

Comment No. 2. *Table 5-8. Particulate Emission Factor (PEF). For the future worker, the Army has chosen the default value for PEF recommended by USEPA Region 9. This value is 1.31E+9 m3/kg, which corresponds to an atmospheric dust concentration of 0.76 ug/m3. Because of the generally dusty conditions at Fort Irwin, we feel this value is too low. For exposures for the future worker, we recommend using the value shown in Table 5-7 for "normal activity" for soldiers, which is 1.6E+7, corresponding to an atmospheric dust concentration of 61 ug/m^3. The Army should recalculate risks and hazards due to inhalation of dust by the future worker at both sites.*

MW Response: Cancer risk and noncancer hazard estimates recalculated for the future industrial worker assuming a particulate emission factor (PEF) of 1.6E+07 increase slightly, but do not change the risk assessment conclusions. These revised estimates have been incorporated into Table 5-8 of the Final RI Report.

Comment No 3. *Section 7 and Table 5-10, Risk Characterization. We concur with the Army's estimates of risk and hazard for military personnel as shown in Table 5-10. After the dust pathway is recalculated, it is possible that manganese might be an additional risk driver for the future worker. We object to the Army's characterization of the concentrations of inorganic chemicals of concern (COPC) as falling "generally within the background range". If this were true, these metals would not be COPC. Stating that these risks fall into the "risk management range" would be adequate for this document. If the Army wishes to compare ambient and site risks, then do so quantitatively.*

MW Response: The revised chemical-specific HQ estimate for manganese, and the total site HI, for the future industrial worker based on a PEF of 1.6E+07 are both below 1.0. These revised estimates have been incorporated into Section 5.5.2 and Table 5-10 of the Final RI Report.

Text in Section 7.1 has been revised to remove the comparison of the arsenic

and manganese concentrations to California background soil levels.

Comment No. 4: *Section 6, page 6-1, Ecological Risk Assessment: At our site visit on 23 March, we concluded with the Department of Fish and Game that Sites FTIR-32A and FTIR-39 did not contain suitable habitat for the Mojave ground squirrel. The sites consist of very little soil on expanses of bare rock. Risks to other indicator species were found to be insignificant in prior screening risk assessment. Therefore, we concur with the Army that these sites pose no risk to non-human species.*

MW Response: Concur.

REVIEW COMMENTS ON:
Remedial Investigation for Sites FTIR-32A and FTIR-39
NATIONAL TRAINING CENTER, FORT IRWIN

From: United States Army Corps of Engineers (USACE)

Comments from Kee Chan, USACE, Dated 28 August 2000.

GENERAL COMMENTS

Comment No. 1: *The Draft is very concise and thorough in discussing human health and ecological risk assessments. This reviewer agrees with the author's recommendation that no further action is required in regards to human health and ecological issues.*

However, this reviewer is concerned that the draft does not discuss about any documentation of the data having been validated or assessed. This deviates from the standard USACE and EPA data assessment protocols.

MW Response: Text has been added to Section 5.1 to explain that the Data Summary Report (Montgomery Watson, 1997) contains the analytical data results and the data validation reports used in this RI. No additional samples were collected during this RI.

SPECIFIC COMMENTS

Comment No. 1: *Page 1-1, Paragraph 1: Please attach a copy of DTSC recommendation.*

MW Response: The DTSC recommendation is included in the Site Investigation Report for Sites FTIR-32A, FTIR-38, FTIR-39, and FTIR-40 (Montgomery Watson, 1998). It is in Appendix D, DTSC Comments from John Christopher, Comment #6.

Comment No. 2: *Page 3-1, Paragraph3: What is "TOpC"?*

MW Response: "TOpC" stands for "Technical Operations Center". This was first defined on page 1-2, Section 1.2.1, paragraph 1. This acronym has been added to the Abbreviations and Acronyms list in the table of contents.

Comment No. 3: *Page 5-23, Paragraph 1: Replace "a b" with "a".*

MW Response: This change has been made.

Comment No. 4: *Page 5-29, Paragraph 3:*

1. *Replace, "TPH" with "TRPH" because the latter was the test performed, according to Table 5-2. Note: Methods 8015M (TPH) and 418.1 (TRPH) are two different tests.*

2. *What were the subsurface depths?*

3. *Who collected and analyzed the samples? Where is the data? The results should be depicted on figure.*

4. *Was the data validated and assessed? Where is the data validation and/or assessment report?*

MW Response:

1. This change has been made.

2. Text has been added to explain that the Data Summary Report (Montgomery Watson, 1997) contains the analytical data results and the depths at which the samples were taken.

3. This information is in the Data Summary Report (Montgomery Watson, 1997) See response above and response to General Comment #1

4. This information is in the Data Summary Report (Montgomery Watson, 1997) See response above and response to General Comment #1